BRAIN.

JANUARY, 1883.

Original Articles.

SUGGESTIONS AS TO THE ÆTIOLOGY OF SOME OF THE SO-CALLED SYSTEM-DISEASES OF THE SPINAL CORD.

BY H. DONKIN, M.B. (OXON.), F.R.C.P.

Physician to the East London Hospital for Children, and Senior Assistant Physician to the Westminster Hospital.

Among the most salient facts in clinical medicine and pathology that recent investigation has brought to light are certainly those relating to the differential diagnosis of some diseases of the spinal cord. There is no doubt that clinical study, stimulated and enlightened by anatomical research, has enabled us to discriminate affections which really can lay claim to individuality, and are found to be associated with a definite lesion when examined *post mortem*. Till quite lately, as is well known, many of these affections were classed together under the general term of paraplegia, or myelitis. Though doubtless existing for an indefinite time they have remained unnoticed in a confused mass of morbid symptoms, till, like the statue set free by the sculptor from the marble block, they have come into being through the labour of the clinician and the pathologist. Much more can now be learnt than heretofore of the life-history of this class of affections, and an improved prognosis ought necessarily to follow in the wake of this diagnostic advance.

Yet as we contemplate the progress we have made, we cannot but be struck with our abiding ignorance concerning the origin of these diseases, not to mention the absence of any line of treatment, which, of course, to be rational, must largely depend on ætiology. On every ground this question is of commanding interest to the pathologist and the physician. The post-mortem study of such cases of spinal cord disease as end in death, and can be thoroughly examined, has at present given us but little direct help towards tracing their causation from the beginning. When we thus examine the body the mischief is already wrought; destructive lesions at most are demonstrated to us; though we may be led by a kind of natural inference to suppose some change in the cord, antecedent to the symptoms and to the structural lesion before our eyes.

But in face of these facts, and though the morbid anatomist has made no general claim to teach us anything regarding the ætiology of the diseases to which we refer, it would seem that many are led to regard too exclusively the lesion found in the cord as the primary source of the malady in question, and so incidentally to limit the field of inquiry as to how these special diseases may be brought about. But little attention is paid to the possible ætiological antecedence of peripheral disturbance, or what may be called abnormal function, which, by its continuance, may set up the ultimate change in the structure of the cord, entailing all the morbid symptoms of the established disease.

In speaking of the possibility of functional disorder producing structural change, it must be acknowledged that at first sight the natural order of things is apparently reversed, and the behests of a truly materialistic pathology seem to be too lightly regarded. But it must be remembered that the word "functional," when applied to disease, does not in any sense exclude the notion of a material cause. We mean by the term such disorder as is not preceded by any structural change detectable by our present means of research, and further, because in many cases such disorder tends to recovery, in all probability not often followed by any permanent alteration. In such a case the word "functional," though theoretically pro-

visional in character, has a practical value, and denominates a real class of affections. Such diseases as chorea and the many disorders of function shown by the various forms of hysteria are instances in point. There are, as Dr. Wilks has said, but few forms of organic nervous disease (i.e. nervous disease demonstrably associated with structural change) that do not find their copy among the hysterical. Does not this close correspondence of so-called hysterical or functional disease with disease that is called "organic"—a correspondence sometimes so close that almost the only differentia seems to be that the one tends to recovery, the other to permanent disablement or death—give us at least a hint of some possible common element at work which may underlie in some cases the beginnings of both affections? In other words, may we not conceive of the more central nervous structures suffering from a continuance of undue excitation from the periphery, so that what at first may have been, for instance, a temporary derangement of the circulation, may come to be a permanent alteration in structure due to chronic congestion or inflammation?

In applying this view of a functional origin to some of the system-diseases of the cord, we must bear in mind that it can only be regarded as a suggestion, to be used or not as it is found to be fruitful, and not contradictory to established facts. Even if it be looked upon as a "*vera causa*," it could with difficulty be demonstrated, the morbid procedure being ex hypothesi in progress during life, and causing thereby an impossibility of tracing continuously the working of the primary peripheral disturbance towards the ultimate structural change. But it may be of some use to consider the subject for a while from this standpoint, and to inquire whether this view derives any support from analogical instances, or falls in with established facts or received theories regarding the physiology of the nervous system.

The study of the working of the nervous system, both in health and disease, is confused and hampered at the outset by an enormous difficulty. We have to deal with the functional aspect of the nervous system; with nervous action, or, as we often term it, nervous force—a force of whose nature

we are ignorant, uncorrelated as it is at present, but which nevertheless we must recognise and provisionally describe. The functional activity of nervous structures is therefore quite a different thing to discuss from that of other organs. Dilate as we may upon the action of the nervous system, whether normal or abnormal, we can as yet do but little towards explaining the phenomena we are grappling with. The words we use, such as "nervous force" and, more notably, "discharge" —of late so frequently occurring in writings on nerve-pathology —are at best but analogical and indefinite in character, in no degree explanatory in the true sense of tending towards simplification, or of expression in terms of the known. We are indeed almost obliged to call in the aid of non-physical terms when speaking of some nervous phenomena, so intimately are they blended with factors which, for the sake of clearness, must be called mental. The unknown element in this field of study is too great for us to institute as yet a scientifically consistent nomenclature. We cannot intelligibly express in definite terms the simplest voluntary movements of our muscles, for we know not the nature of the functional relation subsisting between the brain-cells and the periphery; and we can hardly do more than attempt to give a metaphorical account of the higher workings of the cerebrum, or of the well-known processes whereby a so-called mental impression, though involuntarily arising, is followed by an obvious physical result. We are, in fact, constantly confronted in our study of nervous phenomena with the deepest problems of life, the relation of brain and mind, and we must, in our ignorance, of necessity confuse (albeit with conscious acknowledgment) the terms of psychology and physiology. This unexpressed vital factor is an ever-present "crux" in our endeavours at explanation, and a defiant enemy of post-mortem discovery.

It is necessary to bear in mind this difficulty and source of inaccuracy in our terminology, when listening to any ætiological speculation in nerve-disease. If physiology gives an uncertain sound, pathology may be expected to do the same, or, at least, must not be forced, if it be heard at all, into narrower and stricter limits of diction than those granted to physiology. It may be legitimate and even useful to assume

an undemonstrated pathological process as a tentative explanation of any given phenomena not otherwise understood, provided such a process be not contradicted by accredited knowledge, and be in nowise deemed to be established just because, if assumed, it appears to be adequate to the production of its alleged result. This justification there seems to be for inquiring as to how far a functional origin might serve to account for certain diseases of the cord (as, for instance, locomotor ataxy or spastic paraplegia), without contravening undoubted facts. For there is an especial difficulty while considering these diseases from the standpoint of the *structural* lesions observed, in the view of the *primary* occurrence of anatomical change in certain *functional* or *physiological* paths.

The pathological aspect of these affections to be discussed, while appearing at first sight out of harmony with the ordinary method of study, seems more reasonable when the complexity and peculiarity of the nervous system is borne in mind. The arrangements and mutual relation of the elements of the nervous system, the elaborate structure of the nervous centres, as well as the question of the action and reaction of the individual and the environment, in the development of the higher centres and functions, may all, to some extent, bear on the pathological study of nerve-disease. Perhaps we are all too apt, especially in medical practice, to regard the human individual in his aspect of independence rather than as an integral part of the universe, and to forget that the manifestations of both normal and abnormal action in man may be partially explained by reference to how and whence he came, and to the influence on his organism, both through heredity and present action, of impulses from the external world.

The current manner of speaking of nervous phenomena tends to present a one-sided, or rather, probably, an incorrect view of the mutual relations in structure and function of the several parts of the nervous system. When we speak of the nervous centres, meaning more especially those in the brain and cord, we are apt to regard them in our minds as in every sense antecedent to and causative of, both retrospectively and actually, the rest of the nervous system and its phenomena. We use, for instance, the term "nerve-force" (though more as an exigency of nomenclature than as conveying a very definite

idea), and almost irresistibly regard the nervous centres as the "fons et origo" of this force. We tend to represent the completely developed nervous system, as we see it, as antecedent to the display of nervous action. Generally speaking, in relation to the individual, it is sufficient for all practical purposes to describe the nexus between structure and function in the ordinary manner, and to regard all function or activity as dependent for its existence and character on the structure of its proper organ, without regard to the evolutional history of the organ itself. For this mode of regarding it is, as a rule, scientifically accurate *as regards the individual*. We may thus rightly infer a primary alteration of structure in various organs of the body from an ascertained abnormality of their function, and we find that definite lesions are constantly followed by definite sets of symptoms. Ample instances of this are afforded us by the study of visceral pathology. But when we come to the nervous system, the matter and manner of investigation are not so simple. Instead of a comparative homogeneity and simplicity of structure and function, we find the greatest complexity and diversity. The differentiation of structure is here so great, and the nature of the functions so various and, as it were, unique, their study deriving so little proportionate help from direct experiment, that we must carefully consider the subject in all possible aspects before using a terminology and method of description which cannot be without its influence on future work, and may possibly obscure, instead of tending to advance, the true understanding of the problem.

It is the developmental aspect, then, that we cannot afford to ignore in relation to the physiological and pathological working of the nervous system. For here especially does there seem to be some indication of certain practical bearings on the individual of developmental influences, or, in other words, a different ratio of value to obtain between the hereditary and acquired elements of the organism. Though the hereditary factor may still be quantitatively preponderant, the acquired factor asserts itself visibly, and is obviously of great and characteristic importance.[1]

[1] This is clearly seen of course in those so-called psychical functions which are more especially characteristic of the human species. The notable modification of an individual by circumstances, or the irresistible effect of what Spencer calls the

But little is known by direct observation of the details of the development of the nervous system in man. Certain main points, however, regarding the evolution of nervous structures generally should be borne in mind. And chiefly does it concern us here to remember that "embryological evidence shows that the ganglion cells of the central part of the nervous system are originally derived from the simple indifferentiated epithelial cells of the surface of the body; while the central nervous system itself has arisen from the concentration of such cells in special tracts."[1] Complexity of the nervous system is a characteristic of later evolution in the animal series, and some of the structural arrangements of the nervous centres existing in the higher animals are not found in the embryo, or even soon after birth, but increase in a demonstrable manner during the life of the individual. Apart from the probable fact that the sharp distinction between the white and grey matter is a feature acquired in the higher vertebrata, we know that the grey matter of the cerebrum does not exist in the human embryo, and that its thickness continues to increase through a long period of years. There is then an increase of structural complexity during the period of active cerebral life. Remembering, then, the admitted influence of external stimuli on the elaboration of the nervous apparatus, and its probable effect on the individual, we cannot but think that some different consequences from what we see in the case of other organs may be entailed by this view on nervous pathology. The lung of the new-born infant is essentially similar to that of the adult; and preparations of its liver or its kidney present no structural difference from those of the man. But when we consider that some of the most pre-eminent functions of the human individual—the functions of the nervous system which

super-organic environment, is a striking example. In illustration of this it may be remembered, as quoted in another context by Mr. Henry George in his recent work on 'Progress and Poverty,' that the special form of articulate speech with which we express our ideas (i.e. our national language) is entirely the result of impressions made on our individual brains, though the hereditary capacity for articulate speech is one of the most important *differentiæ* of our species. A child of any country could learn with equal ease the language of all, if placed in similar native surroundings.

[1] See Balfour's 'Comparative Embryology,' vol. ii. ch. xv.

tends so largely to the differentiation and predominance of man in the animal series—are correlated with structural arrangements which are not complete at the beginning of life, it appears most probable not only that modifications and interferences with the developing organism may take place, but also that their significance, occurring as they do in such important material, will be proportionately great. If nervous structure becomes organised abnormally, we may then expect in turn a more or less permanent aberration of function as a result. The structure of the nervous centres, in fact, is to be contrasted, rather than compared, with that of other organs. The fact, again, of the least stable part of the organism, or that part which is most lately developed, being the first to undergo dissolution or degeneration, is exemplified in the study of the nervous system both in health and disease. The grey matter of the brain in old age tends to waste, and its functions to approximate to those of early life; and the tremors of senility are comparable to symptoms which are referable, before the period of natural decay, to cerebral degeneration.

Similar influences, then, to those which in the normal course of things tend towards the development and complexity of the nervous centres, such as an increasing variety of impulses and stimuli from without, or the multiform contact of the environment with the organism, acting through the peripheric nerves, may in altered conditions produce pathological results. The normal complex nervous centres may be best regarded not as primary originators of "nerve force" and antecedent to its display, but rather as automatic registers of those actions which, at first vaguely exerted, were by frequent repetition conveyed along the "lines of least resistance," and have at last come to be organically marked in definite structure. From this point of view we may roughly liken the nervous centres rather to a calculating machine which, with great complexity of structure, can automatically produce elaborate results, though set in action for every process at a given moment by the simplest means, than, as is so commonly done, to the electric battery of the telegraph system, which is the source of telegraphic phenomena. The calculating machine represents, or, it may be metaphorically said, contains stored up in

its structure the brain-work of the inventor. So the nerve centres are the permanent and complex registers of often-repeated peripheric impressions from the external world, and we may conveniently regard the whole nervous system of man as a reflex apparatus derived from and dependent upon sensory impulses.

It is easy to understand, when these nervous paths are once formed, and closely associated by degrees with their increasingly elaborated central organs, that nervous action may proceed along these paths alone, and that the activity of the central cells in the matured organism is involved in every expression of so-called nervous force. It is as if a central government devised and brought into existence by a community had received in complete surrender, for the purpose of the work being carried on with the greatest efficiency and the least friction, the entire control of the action of that community. Such a government, though really the consolidated expression of many individual wills, would appear at first sight to be the mainspring of the action of all those who had so completely given themselves up to its authority.

On the pathological side, then, this method of presenting the genesis of the nervous centres has its application. As certain peripheric stimuli have gradually contributed in the healthy body (regarding both the hereditary and acquired elements) to a normal organisation of the nervous system, so certain stimuli, not so favourable in their nature, and acting moreover perhaps on an organism with hereditarily weak points, or "unstable" in character, may result in the production of a badly organised nervous structure. And when the nerve-centres become thus mis-organised, their indissolubly associated functions must be abnormal too. Thus, for instance, while we rightly attribute the morbid symptoms which we call a disease of the spinal cord to the structural change observed therein, as proximately causing the symptoms, we may consistently regard the structural derangement of the cord as resulting from primary untoward action from the periphery, preventing or marring the normal course of development. And we may compare the diseased cord which spoils the working of its associated nerves to the calculating machine which is out of

order, and whose maker is not to be found. The complicated nerve-structure, once formed, is absolutely necessary to all action; the contributory constituents have long ago surrendered their initiatory powers. But by a reference to the maker of the machine the cause, at least, of its disorder may be manifested; and thus may the consideration of the development of the nervous centres tend possibly to throw some light on the processes of their derangement.

It is obvious that the more stable or earlier-organised parts of the nervous system, being hereditarily comparatively perfect, may (relatively to the individual) be regarded, both in physiology and pathology, as antecedent to functional action. But the more unstable parts irresistibly remind us of how and whence they came, and to duly understand some aberrations from the normal in the case of nervous disease, we should perhaps have this ever before our eyes, regarding some parts of the nervous centres as dependent for their characters on functional actions in the past, and capable of being modified by its continued influence. For this view is probably true of the whole nervous system when taken in relation with the biological series.

It does not concern us here to trace the first appearance of a nervous system in the animal world, any more than it does to know what order is taken by the nervous link in the vital chain of each individual. But given in man a highly differentiated nervous system, it is impossible but that he will suffer materially from its derangement in any degree. The complexity of its arrangement and the importance of its manifold functions are sufficient evidence of this. By it man is brought into relation with the external world. It is at once the highest in function, and the most liable to derangement of all the systems. Nature's "last work which seems so fair" is open to the evil influences of what seem to be ordinary causes. The highest functions of all may be irreparably damaged by influences which are traceable by inference or conjecture alone, and which reveal no morbid stamp when their associated structures are examined in the light of our present knowledge. But we are none the less sure that, though it eludes our search, some "havoc in the tender cells" of the

brain has yet been wrought. And coming to subordinate departments of the nervous system, less subtly constituted than those whose structure is involved in the manifestations of insanity, we should not be surprised to meet with cases of disease in which some peripheral disturbance, owing to interference with the nervous paths, had caused an alteration, either temporary or permanent, of the central nervous structures—an alteration sometimes to be detected and demonstrated by comparison with the normal structure of healthy development.

Again; as in the individual the most recently differentiated and most complex structures are the most likely to suffer from undue stimulation, so too we might expect to find certain cases where the organism had a special liability to such derangement, or an hereditary tendency to fail on the side of the nervous system, even on the provocation of such slightly undue stimuli as might be difficult to detect or impossible to demonstrate. We may have to deal with an inherited "nervous constitution," the vagueness of which much-used and much-abused term may be to some extent diminished by the present point of view. That a certain so-called "neurotic" tendency is found in certain families is admitted by all clinical observers, and is in accordance with what we think we know of the physiology of the nervous system. There is a further point, again, which these considerations illustrate. Although the neurotic tendency is often inherited, it is not a matter of common observation that this is the case with special neuroses or definite "nervous" diseases. We may have many different morbid expressions of nervous action occurring in the same family, such as epilepsy, or chorea, or perhaps various forms of paralysis. And hysteria in its multiform aspect (though no more multiform than might well be expected if we duly regard it as want of proper systemic nervous control) is often a notable mark of many closely-related persons; but the form which the hysterical display shall take is largely determined by the physical and psychical surroundings of each individual case, or, in other words, by the nature of the untoward stimulus.

It is of course obvious that the application of this point of view to the ætiology of any of the so-called "system diseases"

of the cord is beyond the region of demonstration. We are at once confronted with the difficulty or impossibility of showing how such post-mortem lesions as are observed in the cord can have resulted in the manner suggested; and with the further obstacle, that in many cases we cannot prove the existence of any obviously undue functional action at all. The hypothesis must be regarded as merely tentative, perhaps to be completely discredited by future knowledge, and must be granted the full measure, due to all hypotheses, of *à priori* assumption. Thus, in the case of any given specialised disease of the cord, although in some instances obvious functional disturbance, whether in a presumably predisposed individual or not, may irresistibly direct our attention to its probable ætiological importance, yet in many others we can demonstrate no probable exciting cause. Then we may call to mind the inference from other considerations which teaches us the presumable existence of hereditarily unstable nervous structure, and the probable fact of this instability being transmitted more as a tendency than in any specialised form or expression. The patient whose case puzzles us may have been the inheritor of a nervous system thus characterised, and hence requires a much slighter exciting cause (though that cause, be it remembered, must be of a special nature) to bring about an ultimate central change similar to that which, with an obviously great exciting cause, may be presumably found in a better constituted individual. It is because the so-called system diseases, especially those connected with changes in the white columns of the cord called respectively *posterior* and *lateral sclerosis*, or "locomotor ataxy" and "spastic paraplegia," seem so difficult to explain ætiologically from direct observation, and yet from their peculiar course are so interesting and important from this point of view, that some hypothetical discussion of their cause seems excusable. As careful anatomical examination has progressed, so has it come to light that in certain diseases, clinically well marked, there are constant demonstrable post-mortem changes, and, moreover, that these changes take a *physiological* direction, or follow a tract of *special function*. It is the question why this change should start into being in a definite functional region that arrests the attention and de-

mands inquiry; the progress of the change once so started, mainly along the same tract, presenting no great difficulty.

The disease clinically known as *locomotor ataxy* is characterised by the well-known sclerotic change in the posterior root-zones of the cord on both sides; and more recently it has been shown with great probability that a definite set of symptoms, now best characterised by the name of *spastic paraplegia*, but commonly called "lateral sclerosis," is associated with a similar morbid process, symmetrically affecting a part of the lateral columns of the cord known as the crossed pyramidal tract. The anatomical recognition of the latter disease does not rest upon a large number of facts; but the clinical picture is very complete. The post-mortem change cannot be often seen in the unmixed cases, as they run a long and indefinite course; but there is at least one case on record[1] which has almost all the value of an "experimentum crucis."

It is this physiological or functional direction taken by the morbid process that suggests some explanation other than the haphazard occurrence there of a primary inflammatory or degenerative change. The peculiarity of the localisation of the mischief would meet with some explanation if it could be admitted as possible that some morbid influence, or irritation, —call it by what name, or express it how we will—working molecularly, or otherwise (for in our ignorance it matters but little what metaphorical term we use), can be transmitted from the periphery to the centres, and bring about the ultimate changes which are now recognised. We could then conceive with some consistency that an over-excitation of motor or sensory functions of certain parts might account for observed changes in the tract or centres in immediate connection therewith. In such a tract or centre the motor apparatus is gathered, as it were, into a focus, and might represent in its morbid state the result of untoward stimulation from the periphery, just as in its healthy state it is the result of a series of normally conveyed impulses.

Have we, then, any justification in fact or analogy for the

[1] A case brought by Dr. Dreschfeld, of Manchester, before the Pathological Section of the recent International Medical Congress. See 'Transactions of Congress,' vol. i. p. 407.

hypothesis of a functional origin for these definitely marked diseases? Considering the peculiarities which specially mark the nervous system, it cannot be expected that we shall find any closely-related facts in illustration of this hypothesis, which for its chance of life must mainly rest its defence on the absence of facts in positive opposition to it; but the following considerations appear to have some relevancy.

The clinical study of nervous diseases suggests several examples of more or less permanent disorder, which may find at least a not unlikely origin in functional disturbance. Leaving out of the question for the moment the existence or not of structural change demonstrable *post mortem*, we can call to mind in this context the innumerable examples of confessedly physical states, brought about and modified by influences acting on some part of the nervous system; these results being tangible and demonstrable, though the so-called central organ whose action takes part in the phenomenon must certainly be looked upon as being affected in a very different way from what we are in the habit of calling "structural" change. The varied field of the so-called action of the mind on the body is an illustration of this — where often a stimulus to some of the peripheral nerve organs, applied either directly or indirectly through the medium of memory of analogical states in the past, or *mental action*, causes an altered physical state easily recognisable by the observer. Thus a word, or a thought, will cause a blush; the blush being proximately dependent on disturbed vaso-motor mechanism. The same condition may be brought about by the purely physical experiment of dividing a nerve. It is not stretching a point too far to surmise that a constantly repeated stimulus of the first order, could it be attained, would bring about a state of more or less permanent congestion of capillaries.

The influence of various sights, smells, and even thoughts, on appetite and digestion is well known. Undoubtedly, a change in the physical state of the organs concerned causes the nausea and indigestion that are so well known, in certain kinds of temperaments, to result from the untoward stimuli alluded to. Atrophy of organs, of course, is obviously due in many cases to their functional disuse; while hypertrophy is a

consequence of their over-use. Hypertrophy of the heart is known to result from an undue strain upon this organ, dilatation being probably the first stage in the process. But as a further result we often find fatty degeneration, accompanied by an increase of the connective tissue. Here we have an example of structural change occurring in ascertained lineal descent from functional over-work. In some cases, as in pregnancy, the cause of hypertrophy of the heart being not continuous, or, in other words, the functional demand being intermittent, the structural change is not permanent, and does not go on to degeneration; the whole process here may be called physiological. But in most other cases the cause is more continuous, and the morbid result is permanent. Thus we see how narrow a line may divide a physiological phenomenon, demonstrable by even structural change, from a pathological process which we call disease.

To come now to what is more proper to the subject in hand—the diseases referable to the changes in the spinal cord. The most common and best-described example of the system-diseases of the cord is the malady known as locomotor ataxy, or tabes dorsalis, which, by ample post-mortem observation, has been connected with sclerosis of the postero-external columns of the cord. As the observation of this affection has been made with increasing attention, so the manifold nature of its symptoms has become more and more obvious. The symptom of ataxy which, when the disease was first described, occupied such a prominent position, is now rivalled or outrivalled in importance by a host of sensory and even of cerebral disturbances which must necessarily be taken into account when considering the probable origin of the affection. Some authors, as is well known, regard the disease as essentially one of the sensory system, including the symptom of inco-ordination. Doubtless in most cases sensory symptoms of a pronounced character mark its onset. The prominence of the sensory symptoms in locomotor ataxy has lately been especially insisted on by MM. Pierret and Rougier of Lyons, whose writings on this subject are very suggestive.

Various exciting causes of this disease have, from time to time, been put forward. Before the post-mortem condition of

the cord had been so carefully studied as it now is, a theory of the functional origin of tabes enjoyed a much larger share of attention than at present. It was a wide-spread opinion that excessive sexual intercourse was its frequent or general antecedent. This opinion, not traceable to any theoretical bias, but presumably to some extent founded on a basis of observation, has almost disappeared from the schools, owing to the proved connection of the symptoms with structural disease of the cord. This definite sclerotic change, whether primarily inflammatory or not, and wherever the exact histological seat of its origin may be, now alone arrests the attention; and every known cause of such a change is called into requisition to explain it. It is not here my intention to discuss this matter in detail; but this much may fairly be said, that no reasonable theory of the origin of this definite and localised change in the cord has as yet been started. Syphilis, that inexhaustible cruse of ætiological oil, may be regarded as having been weighed in the balance of causation and found wanting. Recourse was doubtless had to it, with that natural longing for finality and logical rest which characterises the minds of most of us; but both on account of the wide prevalence of syphilis, and the known habit of its morbid deposits, *which do not make choice of functional tracts*, its supposed causative *rôle* in locomotor ataxy cannot be at all satisfactory to the serious inquirer. The suggestion, in order to remove a difficulty from the syphilitic theory, that the morbid process may have been first diffused over the cord, and afterwards become localised in the posterior columns, is somewhat gratuitous, and certainly quite out of accord with the clinical history of most cases of ataxy.

But though the origin of this disease is confessedly obscure, there would appear to be some points of probability in the older notion that it may lie in what is to the affected individual relatively excessive sexual excitement. The far greater frequency of tabes in men than in women agrees with this view; and a very strong impression prevails both among practical medical men and others that its causation is of this nature.

I am unable to give adequate details in support of this

position, having been in the habit, in common probably with many others, of to a great extent ignoring this point in cases which have from time to time come before me, owing to the prevailing influence of modern teaching. But as I deprecate these remarks being regarded as claiming any demonstrative value, or as being anything else than barely suggestive, and as I am not attempting here to discuss fully the pathology of tabes, this omission is less inexcusable than it may seem. Long before I was led to theorise at all on the subject, I had difficulty in rejecting from my mind the probable influence of sexual intercourse in the production of this malady. Some cases that I have in my memory strongly impressed me at the time of seeing them; the patients themselves, without previous knowledge of the name or nature of their disease, being confident of its cause, and stating their opinion unasked. These patients' accounts of their excesses were not equivocal; one man especially, with a previously clean bill of health, giving a history where, during two or three years, increasing indulgence had been accompanied by weakness and darting pains in the legs, these symptoms having been relieved by occasional short periods of abstinence. But a typical and rapidly progressive case of ataxy soon developed itself as a sequence, if not as a result, of what all would admit to be a course of extraordinary venereal excitement.

A case is also within my knowledge of a young man of unrestrained desires, who says that after frequent and excessive sexual intercourse he often suffers from symptoms which appear to be temporary attacks of inco-ordination. A commoner complaint, however, after undue indulgence, is that of neuralgic pains in the extremities, more especially in the legs.

It may be said here that Romberg, one of the earliest students of this disease, taught that loss of semen, when combined with that hyper-stimulation of the nerves to which sexual abuses give rise (i. e. to the exclusion of mere spermatorrhœa), not unfrequently favours the origin and encourages the development of tabes dorsalis.[1]

Now if this malady be regarded as one especially affecting

[1] See Dr. Sieveking's Translation (Sydenham Society) of 'Romberg on Nervous Diseases,' s. v.

the sensory system, may not some little light be thrown upon it by supposing the first step in the morbid process to be due to abnormal stimulation of sensory organs; and, assuming for the sake of argument the frequency of relatively excessive sexual intercourse as a forerunner of tabes, may we not reasonably consider the intense and widely diffused sensations accompanying the sexual act as an adequate source of disturbance of an hereditarily unstable, or in some cases even of an approximately normal sensory apparatus, provided that it be admitted that structural alterations in the cord may result from prolonged peripheral irritation? And from this point of view the greater frequency of tabes than of a system-disease of the *motor* apparatus proper derives some little explanation. The possible cause that will be presently suggested for the disease called spastic paraplegia, or lateral sclerosis, viz. over-work of the muscular system, is one that might be expected on this hypothesis to be less frequently active. For the natural demands for action on the motor apparatus are more constant than those on the sexual organs, and in a normally constituted cord we should look for a greater margin of functional activity to be allowed to the motor tracts without risk of over-stimulation taking place.

It may at least be said that this view of the possibility of a peripheral origin of the disease known as tabes seems to harmonise to a considerable extent with all the characteristic clinical facts. I am fully aware that the opinion of many experienced, and deservedly high, authorities on diseases of the nervous system is opposed to any close connection between tabes and the hypothetical cause under discussion. And were I defending a thesis, instead of inquiring, I should not venture to criticise existing views without bringing at least an equal amount of experience and labour towards the solution of this question. But it may be pointed out that just as in chorea, as ably argued by Dr. Sturges in his recent works, the apparently ordinary emotional causes which may produce it are necessarily often overlooked, so, in this instance, what is really relative excess of sexual indulgence might well be passed unnoticed, quite apart from the number of cases where it might be suspected, but purposely denied Of course if this sequence

of events could be satisfactorily substantiated, the question of the causation of the disease would not be set at rest entirely. But on the principle, "Causæ non sunt multiplicandæ præter necessitatem," this hypothesis, serving to cover a large number of cases, would deserve at least a front rank in the investigation of the matter.

A few words may be said here on the arguments used by those who, partly from certain clinical data, partly from theoretical reasoning, deny the sensory factor in the production of inco-ordination, and whose position would, by implication, be opposed to the probability of the origin of the entire disease in disturbance of the sensory apparatus by peripheral irritation. It would seem that the use of the term co-ordination often causes and implies considerable mental confusion. Because a certain tract of the cord is found to be diseased in cases characterised by a want of co-ordination, the minds of many are satisfied with the conclusion that this tract is in some special way the seat or the conductor of "co-ordinating impulses." Now apart from the distraction which has been caused by the pathological study of locomotor ataxy, the question of a special seat or tract for co-ordination in the cord would never have struck the philosophic neurologist. Physiology tells him nothing of it; but, rather than suggest a search for it, would lead him to regard co-ordination as only a functional aspect of the nervous system. Co-ordination simply means the harmonious working of the nervous system as expressed in muscular action, whether considered as a whole or in its parts; and is implied in the very notion of a nervous *system* at all. That this harmonious working is dependent on the proper conduction of afferent and efferent impulses in connection with organised centres, or in other words implies a certain relation between the sensory and motor apparatus of the nervous system, would appear from a physiological point of view fairly obvious; and an impairment of co-ordination, or want of harmonious action in any part, would be referred to derangement of part of the apparatus involved, causing a want of proper conduction of either sensory or motor impulses. A special tract for the conduction or localisation of "co-ordinating" impulses is as little wanted in

pathology as it is hinted at by physiology. But those who argue against the influence of the afferent factor on the production of the *ataxia* of tabes, regarding it purely as a motor morbid symptom, seem at least unconsciously prepossessed with the idea that some special seat must exist for what they appear to treat as the co-ordinating "function," omitting in thought to analyse it into its simpler elements. Forgetting moreover, apparently, the fact that such a localisation has been suggested only by the observed connection between *ataxia* and disease of the posterior columns, and bearing in mind that physiology has as yet given only sensory functions to those parts of the cord, some have endeavoured to find in the lateral column the true seat of what they regard as co-ordination, believing it *must* have a seat somewhere; so that, on this showing, the well-known and accurately defined lesion in the posterior root-zones of the cord does not, after all, account for one of the most important symptoms of the disease in question.

The clinical evidence called in to support this view is based on the quotation of cases where inco-ordination is observed without loss of sensation; or where anæsthesia takes place without ataxia. Quite apart from the fact that the cases are, to say the least of it, very rare and questionable where some degree of anæsthesia does not obtain in connection with ataxia, the confessedly difficult subject of the due investigation and valuation of the symptom of anæsthesia should prevent us drawing any very important conclusion from the little we know of the matter. We learn but very little from direct physiological experiment on sensations, and especially of sensory conduction in the cord. The subject is beset with obvious difficulties; and though investigators speak with almost one voice regarding the motor functions, there is palpable discord among them respecting sensory phenomena. But when we remember (to quote from Foster's work on Physiology) "that an afferent impulse passing along an afferent nerve may in certain cases, simply produce a change in our consciousness, unaccompanied by any visible movements, and in other cases may give rise to reflex movements, or modify existing reflex or automatic action without causing any change

in our consciousness," we shall at least see that it is premature to deny the afferent or sensory factor in the production of inco-ordination, on the strength of such cases as we have alluded to. This question is argued at some length by Professor Erb, in his article on "Tabes," in Ziemssen's 'Cyclopædia,' where I think, in his advocacy of the exclusively *motor* nature of the ataxic symptoms, there is betrayed the confusion I have referred to concerning the *word* co-ordination, and this partly gives a bias to his arguments and quotation of cases against the possible part played by the sensory side of the nervous system in the production of inco-ordination. The consideration of this question illustrates the danger there is in attempting to make physiology too soon—to build up a physiological theory on pathological facts or clinical observations alone. The lines of physiological and clinical investigation, though often giving mutual help, must be kept distinct; the inferences of the latter being never looked upon as the dicta of the former. In the case we have been glancing at we have scientific warrant in maintaining only the *concomitance* of certain symptoms with a certain observed change. This fact is established. But we may not thereupon invent a function to plausibly occupy an insufficiently explained structural locality. This fallacy and confusion, although confessedly not put forward as a theory, or baldly stated by any writer, nevertheless seems to lurk deeply in many discussions on the subject.

To take now another example of the system-diseases of the cord, which may serve on the motor side as a counterpart to tabes on the sensory side. The disease now known as *primary lateral sclerosis*, or *spastic paraplegia*, must be regarded, owing to the admirable work of Professor Erb, as a clinical entity; and, though in all probability less common than tabes, is being recognised with increasing frequency. It is true that the evidence of the associated cord-change rests upon very few observations; but taking into consideration what has been indubitably made out, and what is known, moreover, regarding other forms of sclerosis in the lateral columns, in conjunction with the concomitant symptoms observed—this disease must challenge the attention of those interested in the ætiology of

the system-diseases of the cord. It is believed that certain tracts symmetrically placed in the lateral columns of the cord are especially occupied in the conduction of motor impulses towards the periphery; the normal conduction of these impulses implying, too, the control, or exhibition, of reflex action. Difficulty of motion, stiffness of the legs, and an exaggeration of the reflexes, are the prominent clinical symptoms connected with this form of sclerosis. Is there any clinical justification for supposing that the cord-change existing here may be secondary to undue stimulation of the motor apparatus: granting, for the moment, the possibility of a pathological effect being wrought on the concentrated nerve masses of the motor tract in the cord by undue irritation of the motor nerves of the periphery? Certain points, both negative and positive, seem to favour such a supposition, though they may be not sufficiently obvious to claim recognition. There is no theory, even vaguely accepted, as to the ætiology of this affection, definite as it seems to be. The syphilitic explanation has not been seriously broached, and by most authors is rejected, to fall back on "cold," possible injuries, and such-like general hypotheses—so general, as almost to defy either proof or disproof. One positive point, however, is noticed by those who have written on this disease: it occurs comparatively frequently in men—and in muscular men. It is perhaps in this class that we may expect to find the material for a disease hypothetically producible by excessive demands made on a possibly unstable nervous apparatus: for here it is more likely that muscular overwork might take place, and the possessor of the strongest muscles is not necessarily the subject always of the most perfect nervous control. It must be remembered, too, that this disease, anatomically characterised by sclerosis, most marked in the lower part of the cord, begins generally in the lower extremities, which are more liable to continuous overwork than other parts of the body. In some cases of this disease, moreover, excessive action of the muscles can be clearly established. One case especially,[1] which has lately been under my care, may, I think, be taken as supporting this view. It is that of a young acrobat or contortionist,

[1] See 'Brit. Med. Journal,' Dec. 9, 1882.

aged twenty-one, who about eighteen months ago began to complain of weakness and tremulousness of his legs. When he first came under my observation he had not developed ankle clonus or the spastic gait, and the knee-phenomenon was not markedly exaggerated. With rest he made some marked improvement for a time; but he again took to his work, and gradually became, what he is now, a typical specimen, as far as clinical observation alone can demonstrate, of a "spastic" paralytic. For six months previously to June in the present year he had been performing, against my advice, literally nightly in Berlin and Paris, being obliged more and more to favour his legs by confining most of his "business" to his arms. This youth had been an acrobat ever since four years old. With the exception of having been a "delicate" child, his health appears to have been very good up to the time of his legs beginning to fail. The rest of his family were always strong, and are following the same occupation as himself. I satisfied myself without doubt that he had not had syphilis.

A case of this kind irresistibly turns our attention to the possibility of its functional origin, though of course much more observation is wanted. Taking into consideration the history of the case, the mode of its onset, the amelioration of the early symptoms by rest, and the extreme difficulty of suggesting any other plausible explanation for this typical case, it may be said that the hypothesis of its functional origin covers the facts before us; and, provided it be not shown to be contrary to others, may possibly, if tentatively held, with full readiness to drop it in the light of further knowledge, be found of some service in the study and treatment of paraplegic symptoms.

Other affections ranked under the head of system-diseases of the cord I do not propose to discuss here. Progressive muscular atrophy, and the disease long known under the name (now proved inaccurate) of infantile paralysis, whose morbid anatomical concomitants are localised in the grey anterior cornua of the spinal marrow, present some differences in character from the affections connected with lesion of the white matter that we have been discussing. The lesions here

do not take a physiological course in the disease known as polio-myelitis anterior; and the existence of a primary inflammation, especially affecting the grey matter of the cord, and occurring, as it is believed, in the cervical and lumbar enlargements, is not out of harmony with the clinical facts or the anatomical conditions of the case. The chronic change in the anterior cornua observed in progressive muscular atrophy, corresponding as it does in position to the entry of the nerves supplying the affected muscles, is not contradictory to the theory of the functional origin of the disease. That the mischief begins in the periphery is held by several observers, both clinical and anatomical, and many facts in the history of these cases seem to favour this point of view.

Certain affections of more or less permanence, characterised by paralysis or spasm, but which are not as yet connected with any observed central change, may be here alluded to, as apparently of functional origin, and illustrative of the point under discussion. The frequent great chronicity, for instance, especially when not sufficiently regarded at the outset, of scrivener's palsy, or writer's cramp, would lead us to suppose that some central change had taken place. The marked failure of co-ordination seen in this disease seems to imply some kind of disorganisation of the central structures immediately concerned in the previously harmonious action of the muscles. Yet the apparently functional origin of such an affection as this is very striking, and widely believed in. There is, moreover, reason to believe that it is most often seen in members of "neurotic" families.

The prognostic and, possibly, the therapeutic aspect of this method of regarding some of the system-diseases of the cord deserves perhaps a few words in conclusion. The obvious physical result of overwork (whether the overwork be regarded as absolutely primary in causation or not) in such a disease as scrivener's palsy calls loudly on Rest for its cure or relief; and if taken in time, the disease frequently yields to this rational treatment.

With respect to the prognosis, and perhaps the prophylactic treatment, of spastic paralysis and tabes, a possible hint may hence be derived. The established disease of course implies

the structural central change; and treatment here can do little or nothing. But the study, when possible, of early untoward symptoms which, though known often to precede the well-recognised disease, yet, according to the hypothesis of functional disturbance, may not portend the necessary sequel of the full-blown malady, might help in some cases to prevent the hopeless and dogmatic diagnosis that is in danger now of being given, even on the strength of a single, and especially a newly-observed, symptom; and perhaps in some few cases might hinder the occurrence of the incurable malady. It is possible that in these days of minute and microscopical research we may sometimes fail to hold ourselves off, as it were, and take a look at our facts from a little distance. To get a true notion of our object we must vary our focus and our points of view. And just as the clinics and pathologics of pulmonary consumption are in some quarters regarded, with apparent satisfaction to the mind, as already entirely revolutionised by the discovery of the tubercle *bacillus*, and the accumulated experience of past observers in the field seems to be overlightly, or at least prematurely, set at nought, so in neurology the recently-studied changes in the cord may tend to engross the attention of observers too entirely as ultimate facts, and thus perhaps prevent us from reaping the advantages that sometimes result from merely changing our point of view.

DEAFNESS IN HYSTERICAL HEMIANÆSTHESIA.

BY G. L. WALTON, M.D., BOSTON, U.S.A.

AMONG the perversions of sensibility to which hysterical patients are subject, that of hemianæsthesia is one of the most common, and the study of its various phenomena well illustrates the fact, that this disease is as subject to given laws and as worthy of scientific investigation as any other; a fact which, if not at present universally accepted, is rapidly becoming so, thanks in great measure to the efforts of Professor Charcot and his pupils. It is at his suggestion that I have examined the hearing of the patients (numbering thirteen) with this affection in the Salpêtrière, the result of which examination it is the immediate object of this paper to report.

Before, however, entering upon this branch of the subject, it may not be out of place to devote some space to the general characteristics of the symptom under consideration. A knowledge of these characteristics is especially important, inasmuch as the diagnosis must sometimes be made between hysterical hemianæsthesia and that due to an anatomical lesion.

Hemianæsthesia is one of the more common of the lasting symptoms exhibited between the attacks by hystero-epileptics, to which class belong the majority of the cases examined, all conforming more or less closely to the classical type. There exists in these cases a loss of sensibility, occupying the whole of one side of the body, generally limited sharply by the median line, though not always so well defined, as for instance in the case reported by Féré,[1] in which a sensitive spot, the size of the hand, was found behind, between the dorsal and lumbar regions, and another of similar size near the navel. The

[1] "Notes pour servir à l'histoire de l'hystéro-épilepsie;" 'Archives de Neurologie,' Nos. 8 and 9, 1882.

anæsthesia may be complete, involving loss of sensation of touch, pressure, heat, cold, and pain; or it may be incomplete, in which case analgesia, with or without thermo-anæsthesia in the more common variety. The anæsthetic side is generally paler and colder than the other, and blood flows less easily from the prick of a pin or other instrument. This condition of ischæmia, which is, however, rarely found existing to such a degree, can explain, after a certain allowance for exaggeration, the so-called miraculous cases in the Epidemic of St. Médard, in which strokes of the sword failed to produce hemorrhage.[1]

The anæsthesia is sometimes limited to the integument, and sometimes extends to the deeper parts, muscles, bones, articulations, mucous membranes. The abdominal viscera, however, do not seem to share in the anæsthesia; on the other hand, the so-called "ovarie" generally exists on the affected side; that is to say, an extremely sensitive spot is found in the region of the ovary, pressure on which, if the patient is subject to hystero-epilepsy, can induce one of the attacks, the nature of which has been so carefully studied in the wards of the Salpêtrière, and which has been made the subject of an admirable volume by Richer.[2] Forcible pressure over the same region is capable of cutting short an attack once commenced, and for this purpose various belts have been made, resembling trusses by means of which constant pressure may be applied over the ovary. That this sensitive region really corresponds to the ovary is placed beyond a doubt by the fact that it gradually rises during pregnancy with the rise of the uterus, and falls again after parturition.

The hemianæsthesia is more commonly located on the left half of the body, but it may change to the opposite side in the same patient, together with all the attendant symptoms of special sense which will be presently mentioned. This change may also be effected at the will of the observer by the use of one of the many so-called æsthesiogenous agents. In some

[1] Charcot, 'Leçons,' &c., p. 303. Jolly quotes a characteristic case of ischæmia described by Rosenthal: 'Allg. Wien. med. Zeit.,' 1876.

[2] 'Études cliniques sur l'hystéro-épilepsie ou grande hystérie.' Par Dr. Paul Richer. Paris, 1881. (See review in 'BRAIN,' Jan, 1882.)

cases the anæsthesia occupies both sides of the body, though not always in the same degree, and these cases are particularly favourable for examination with regard to the symptoms of sight, hearing, &c., as the affection of the special senses corresponds in degree with that of the general sensibility. Contractures, when present, occur as a rule on the anæsthetic side, and the motor force, as shown by the dynamometer, is generally found somewhat diminished. The loss of sensation, far from inconveniencing the patients, is often not noticed by them until the attention is called to it, and they sew, knit, and perform other duties without the least disturbance.

With regard to the share which the special senses take in this affection, I cannot do better than to quote verbatim the account given by Féré:[1]—

"In the hemianæsthesia of hysterics the insensibility extends not only to the skin and mucous membrane, but generally also to the organs of sense; the sight, the smell, the hearing, the taste, are equally diminished on the same side. There is generally present, in one word, a sensitive sensorial hemianæsthesia. Still, the organs of sense may be unaffected, or only slightly involved. The eye, for example, which in the case of complete anæsthesia is the seat of the characteristic disturbances, advancing sometimes to total loss of vision, shows more often a concentric retraction of the visual field, corresponding generally with achromatopsia, more or less pronounced.

"The colour-blindness is sometimes complete, but is frequently partial; certain patients lose, for example, only the perception for violet,[2] and in general there is a correspondence between the degree of retraction of the field of vision and the degree of achromatopsia.

"It is a fact which has attracted little attention that the sensibility in general of the eye, the sensibility of the conjunctiva and cornea, bears a close relation to the special sensibility of that organ. The patients whom we have been able to

[1] "Notes pour servir à l'histoire de l'hystéro-épilepsie;" 'Archives de Neurologie,' Nos. 8 and 9, 1882.

[2] "Violet is the colour for which the field of vision is physiologically the least extended, therefore the first to disappear on retraction of the visual field."—Landolt, 'Leçons sur le diagnostic des maladies des yeux,' p. 155.

observe, who have presented no retraction of the field of vision, and no achromatopsia, have preserved the sensibility of the conjunctiva; those who have lost their vision for one or more colours, and who have a retraction of the visual field more or less regularly proportional, have lost not only the conjunctival sensibility, but also that of the cornea. In the latter case, if, while the patient directs the gaze fixedly at some object, one advances a little roll of paper, one sees the eye and eyelids remain immovable as long as the foreign body does not come into the field of the pupil. The oculo-palpebral reflex is obtained quickly if, instead of advancing the object to contact with the membranes, one only advances it part way in the median line. The reflex is then determined solely by the stimulation of the retina, which, though it has lost the power of perceiving colours, can distinguish light and darkness. In the pure hemianæsthetics, or in the total anæsthetics with predominance of one side, one can by the magnet, the static electricity, or the other well-known procedures, transfer the anæsthesia, and make a counter-demonstration with constant results. There is then in hysterical hemianæsthesia a constant relation between the cutaneous insensibility and the sensorial insensibility. And this constant relation exists not only when the anæsthesia extends over the whole of one side of the body, but also when it is more or less limited. When one causes the disappearance of the hysterical anæsthesia by static electricity, one sees the anæsthesia return at the end of a certain time, the return commencing in a localised region, varying in different subjects, and having in general no relation to the distribution of the nerves. In one of our patients the insensibility returns in the first place over a limited zone, which includes the eye, the conjunctiva and cornea, and the sensorial anæsthesia returns with that limited tactile insensibility."

The same author mentions the unilateral deafness which appears in these patients, and states that the general sensibility of the external auditory canal (not the tympanum) varies with the acuity of hearing, just as that of the conjunctiva varies with the field of vision for colours. He draws the conclusion that there exist in the brain sensitive centres,

which supply in common the organs of special sense and the integument covering them.

Jolly [1] remarks that functional deafness is sometimes noticed in severe cases of hysteria, remaining after violent attacks, and combined with other anæsthesia, adding that it may, like hysterical blindness, suddenly disappear, and be replaced by other hysterical symptoms.

Uspenski [2] has lately reported two cases of hysterical deafness, accompanying anæsthesia of the face and external meatus, in one case complicating lesion of the ear, while in the other the physical examination revealed no defect. In both these cases the functional deafness varied with the cutaneous anæsthesia, and in both cases a marked improvement appeared on the application of the galvanic current to the cervical sympathetic.

Gellé has made measurements respecting the distance necessary for distinct audition during the "transfer" of hemianæsthesia, confirming the law that cessation of anæsthesia on one side of the body involves its appearance on the symmetrical part on the other side of the median line.

Urbantschitsch has reported a case of hysterical hemianæsthesia with the "transfer." The subjective noises, and hyperæsthesia for loud tones which existed previously in the non-anæsthetic, were transferred to the anæsthetic ear. The deafness, which was complete, passed meanwhile to the previously hyperæsthetic ear. Before the transfer the strongest galvanic current (40 Siemens-Halske elements) produced on the affected side neither auditory nor optic symptoms, while six elements on the unaffected side produced such violent vertigo as to prevent the establishment of the auditory formula. This relation was reversed by the transfer.

With regard to the genuineness of the anæsthesia in these cases. It must be remembered that the psychological state of hysterical patients is such as predisposes to deceit, in view of which the explorations are conducted at the Salpêtrière with

[1] Ziemssen's 'Handbuch,' Bd. xii. 2, 1877, p. 534.

[2] "Taubheit complicirt mit hysterischer Neurose und hysterische Taubheit." 'St. Petersburg med. Wochenschr.' 1882, No. 8. Reported also in 'Neurologisches Centralblatt,' Oct. 15, 1882.

all the precautions necessary to its detection. That simulation played a part in any of the cases here reported was out of the question, for apart from the various precautions taken, the very regularity of the results precludes deceit.

The inadequacy of "expectant attention" in explaining these results becomes evident on considering the cases in which the loss of hearing for sounds conducted by the bone was lost, while that for sounds in the air was retained. It is absurd to suppose that a number of patients, hitherto unexamined in this respect, on being separately questioned should be found to coincide with regard to this symptom, and in a degree proportional to their several grades of anæsthesia.

Coming now to the special division of the subject, we may divide the patients for convenience into three classes, and consider the hearing in each class separately. The *first class* comprises the patients with *complete* anæsthesia on one side, the other side remaining intact. The *second class* includes the patients with *incomplete* anæsthesia on one side, the other side remaining intact. The *third class* includes those patients with anæsthesia more or less complete on both sides. The degree of anæsthesia in these cases is rarely the same on the two sides.

In the *first class*, as in all, the anæsthesia extends to the deep part of the ear. The anæsthesia being complete in this class, the tympanum may be touched without any acknowledgment of sensation, and without the least reflex movement. In such a case the patient will be unable to tell with the eyes closed whether there is an instrument in the ear or not. Touching the deep parts of the opposite ear in these patients produces the usual disagreeable sensation and reflex movement, generally indeed exaggerated. That the anæsthesia extends to the middle ear is seen by the fact that insufflation by the Politzer air douche produces no sensation in the ear of the affected side. This is a fact to be borne in mind in the examination of these cases, for if the patient's sensations were relied on, the Eustachean tube might be supposed impermeable.

In this class of patients neither the watch, the voice, nor the tuning-fork is heard by the affected ear, and no tuning-fork is

heard on this side when vibrating against the skull. The tuning-fork vibrating in contact with the forehead or teeth is heard *only on the healthy* side, in direct opposition to the cases in which the loss of hearing is due to defective conductibility in the ear, and in which the tuning-fork so placed is heard more distinctly on the affected side.

In the *second class* of patients, those with incomplete anæsthesia on one side, the loss of sensibility in the ear corresponds as a rule to that of the body in general. A common form consists in analgesia with thermo-anæsthesia and diminution of tactile sensibility. In patients exhibiting this degree of anæsthesia, the tympanum of the affected ear may be touched without producing any unpleasant sensation, the touch being only faintly perceived, and being followed by no reflex. The air douche produces a slight sensation. The degree in which the hearing is affected varies within certain limits, but has been found lessened in every case examined. In some of these cases is found a *diminution* in the hearing for sounds conveyed by the *air*, and a *diminution or loss* of hearing for sounds conveyed by the bone. In these cases the watch, voice, and tuning-fork, all of which are heard at a normal distance, or at a distance abnormally great, by the unaffected ear, are heard at a half or a third of that distance by the affected ear, or perhaps only in the immediate vicinity; while the watch or tuning-fork placed on the bone behind the ear on that side is heard faintly, or not at all, when the ears are closed. In these cases the sound of the tuning-fork vibrating on the forehead or teeth will predominate on the healthy side if the hearing for sounds conveyed by the bone is diminished in the affected ear, and solely there if it is lost. The hearing may be approximately or absolutely normal for sounds conducted by the air, and lost entirely for sounds conducted by the bone, as in the case of one patient who can hear all tuning-forks distinctly in the air, and can hear the watch, normally heard at 80·100 ctm., at a distance of more than a metre, while no tuning-fork is heard on the affected side when vibrating against the skull at any point, as long as the ear is closed. In the normal condition, closing the ear, as is well known, intensifies the sounds conducted by the bone.

In regard to the *third class* of cases, in which the anæsthesia is total, the completeness of the anæsthesia is rarely the same on both sides, a common form being complete hemianæsthesia on one side, and analgesia on the other. These cases are particularly interesting as regards the study of the hearing, for the degree of deafness always corresponds to that of the anæsthesia. To illustrate; one patient has complete hemianæsthesia on the left, and analgesia with diminution of tactile sensibility on the right. This patient cannot feel the contact of an instrument with the tympanum on the left; on the right she feels the contact more or less distinctly, but it gives rise to no unpleasant sensation. On the left she hears neither watch, voice, nor tuning-fork, even in the immediate vicinity, and no tuning-fork vibrating against the skull. On the right she hears all with greater or less distinctness by the air, but perception for sounds conveyed by the bone is absent. A tuning-fork vibrating against the forehead or teeth is not heard at all when the right ear is closed, though with the right ear open the same tuning-fork may be heard by the air quite distinctly.

Another patient has complete hemianæsthesia right; incomplete, left. She feels nothing when the right tympanum is touched, but a slight sensation when the left is touched. The air douche produces a faint sensation in the left, none in the right. This patient hears no sound on the right, whether conducted by the air or by the bone. On the left, the watch is heard on contact only. A tuning-fork placed on the bone behind the left ear is heard faintly when the ear is open, and still more faintly when the ear is closed. No tuning-fork vibrating on the forehead or other part of the skull is heard at all.

In this case the hearing, which is completely lost on the right, is greatly diminished on the left, both for sounds conducted by the air and those conducted by the bone.

These two cases will serve to illustrate this class, in which an infinite variety may be imagined, the general character remaining the same.

A noticeable feature in all these cases, and one which throws some light on an important physiological subject, is the uni-

formity with which the deafness for sounds conveyed by the bone exceeds that for sounds conveyed by the air. This is probably due to the fact that the vibrations conveyed to the ear by the air are better adapted for the irritation of the peripheral auditory apparatus than those conveyed by the bone.[1] When then the receptive power of the auditory centres is lessened, as is probably the case in the hysterical patients, the hearing for sounds conveyed by the bone disappears before that for those conveyed by the ear. This enfeeblement of the auditive centres in hysteria is quite analogous to that in old age, in which, as is well known, the perception for sounds conveyed by the bone disappears before that for sounds conveyed by the air, the former being sometimes completely lost before the age of sixty.

Some authors attribute this condition in age to a lessening of the conductibility of the bones of the skull, which is a very improbable explanation, for if the conductibility of these bones for vibrations becomes altered as age advances, it is probably rather increased than decreased with their increasing solidity. The ears, meanwhile, of the large majority of persons undergo such changes as age advances, thickening and indrawing of the tympanum, closure of the Eustachean tube, &c., that comparatively few persons at sixty preserve the normal conductibility of the tissues of the ear. As far, then, as the conducting media are concerned, the deafness for sounds conveyed by the bones should never be expected to precede that for sounds conveyed by the air. When the hearing for the former sounds disappears first, it must rather be attributed to the gradual enfeeblement of the auditory centres in the brain. In the hysterical patients there can be no question of a change in the conductibility of the bones, but all symptoms must be referred directly to the nervous system, and in these cases, as has been seen, the hearing for sounds conveyed by the bone always disappears first. On this question, as on many others in the physiology of the nervous system, these hysterical patients point the way to the most satisfactory solution.

[1] That this is really the case is evident from the simple experience that a tuning-fork, feebly vibrating, is heard much longer through the air than through the bone.

It need hardly be added that the condition of the ear itself may modify the cases to a certain extent, a fact not to be forgotten in the analysis of the cases, for distinction must be made between deafness due to a cessation of function of the auditory centres, and that due to an accumulation of cerumen, or to a chronic catarrh of the middle ear. The examination by osseous conductibility is here of the greatest value, for whereas obstruction in the ear itself intensifies sounds conducted by the bone, such sounds are the first to disappear in hysterical deafness.

The following cases, typical of all, will serve to illustrate the three groups of patients mentioned.

Case I.—Mlle. Bl——, 20 years old. Complete hysterical hemianæsthesia, left.

Senses of *taste* and *smell* lost on the left. With the left eye patient distinguishes only the colour red.

Examination of the ears shows membranes normal. Touching the tympanum is neither felt nor followed by a reflex movement on the left. On the right the sensibility is normal. Politzer air douche not felt on the left.

Examination of hearing.—The patient hears neither the voice, the watch, nor the tuning-fork on the left; the two latter, whether in the air or on the bone. The watch (heard normally at a distance of 80–100 centimeters, is heard on the right at 85 centimeters. On the right the voice, whispered and murmured, is heard at a normal distance, and all tuning-forks are well heard, whether vibrating in the air or against the bone. The tuning-fork placed on the teeth or forehead is heard only on the *right*.

Case II.—Mlle. Card——, 16 years of age. Incomplete hemianæsthesia, left; the sensations of pain and of cold are lost, and that of touch lessened. Retraction of the visual field, left. No achromatopsia. Sense of taste entirely, and that of smell nearly, lost on the left.

Examination of ears showed right tympanum normal; the left indrawn and opaque. Left Eustachean tube impermeable. Touching the left tympanum produces only a faint sensation.

Examination of hearing.—Patient hears the watch at 130 centimeters on the right; only at contact on the left. The

whispered voice (using the word "cinq") heard normally at about 10 meters, is heard on the right at 14 meters; on the left at 20 centimeters. All tuning-forks are heard in the air on the left, but none on the bone. Neither the watch nor tuning-fork placed on the bone with the ear closed is distinguished on the left. *A tuning-fork placed on the forehead or teeth is only heard on the right.*

CASE III.—Mlle. Lim——, 19 years old. Incomplete hemianæsthesia, left.

Special senses.—Patient distinguishes all colours on the right; only the red and green on the left. Retraction of visual field, left. Taste and smell are entirely absent on the left; slightly diminished on the right. V. = $\frac{5}{7}$ right; $\frac{5}{10}$ left.

Examination of ears shows the membranes approximately normal. Touching the tympanum on the left is felt, but produces no reflex movement nor disagreeable sensation. The deep parts of the right ear preserve their normal sensibility.

Examination of hearing.—The watch is heard on the right at 56 centimeters; on the left at 13 centimeters. The whisper is heard on the left at 30 centimeters. The tuning-fork is heard normally on the right, both by the air and by the bone. The tuning-fork placed behind the ear is not heard when the ears are closed. *The tuning-fork vibrating on the forehead or teeth is heard on the right.*

CASE IV.—Mlle. Mig——, 16 years of age. Complete anæsthesia, left; analgesia, right, with diminution of tactile sensibility. *Muscular sense* preserved; *odour* lost on the left, diminished on the right. The same is true for the taste. On the left, sees only the colour red. On right, all colours. Visual field greatly contracted left. V. = 1 right; $\frac{1}{7}$ left.

Examination of ears shows the membranes normal. Touching the membrane is not felt on the left, slightly on the right.

Examination of hearing.—Patient hears the watch on the right at 120 centimeters; on the left, not on contact. The voice is not heard on the left. The tuning-forks are not heard on the left, either in the air or on the bone; on the right they are heard in the air, but not on the bone with the ear closed. The largest tuning-fork, placed on the forehead, is unheard when the right ear is closed.

The second and last cases illustrate a condition not uncommon in hysterical hemianæsthesia, i.e. an unusual acuteness of hearing on the unaffected side. The watch, heard normally at 80–100 centimeters, was heard in one case at 120 centimeters. This acuteness of hearing was transferred, without diminution, to the other ear in Case IV. (in which the membranes were normal) in the experiment to be immediately described.

The transfer.—The fact that hysterical hemianæsthesia, with all its symptoms of special sense, may change its seat, and that by means of certain agents this change may be brought about at the will of the operator, has been known for some time. This change has been known as the "transfer." In 1878 Charcot called attention to the so-called "oscillations," which are merely a continuation of the transfer. Rumpf[1] has demonstrated, by a series of careful experiments with the compasses, that similar oscillations of sensibility from side to side take place under physiological conditions. The oscillations in hysteria have been made the subject of careful study by Richer, the result of whose researches were published in the 'Progrès Médical' (Nos. 46 and 47).

To quote several of his conclusions:—

"III. The oscillations take place in the same way, whether the æsthesiogenous agent be removed or maintained.

"IV. The oscillations exist as well for the special as for the general sensibility.

"VI. The duration of the oscillations is very variable; we have observed oscillations of several seconds, and others of twenty minutes. They may last even longer. One may say in a general way that the oscillations are the shorter and the greater in number, the more easily they are produced.

"VII. When the oscillations are finished, the patient remains, as far as sensibility is concerned, in a state of relative stability. We have observed three varieties. 1. The patient recovers, for some time, total sensibility. 2. The patient remains in a state of transfer, i.e. if she was hemianæsthetic on the left before the experiment, she remains for

[1] "Ueber den Transfert;" 'Berliner Klinischer Wochenschrift,' 1879, No. 36.

some time hemianæsthetic on the right; perhaps one should consider this condition merely a prolonged oscillation of which the end has not been observed. 3. Finally, the patient finds herself in the same condition as before the experiment."

The study of these oscillations is especially interesting in connection with the study of special sense, and observation of the several varieties mentioned above shows that the same rule holds good in all, for the hearing as for the other senses, i.e. that displacement of anæsthesia on one side is accompanied by its appearance on the other, and always to a corresponding degree. Given a patient who hears perfectly by air and bone on the left, but who has complete deafness on the right, after the transfer she will hear perfectly (there being no obstruction in the ear itself) by air and bone on the right, while she will have completely lost the hearing on the left. This condition will change from side to side during the oscillations, until the patient arrives at one of the states of relative equilibrium mentioned by Richer. The sensitiveness of the tympanum will oscillate in a corresponding manner.

Given, again, a patient who has lost on the right perception for sounds conveyed by the bone, but preserved that for sounds conducted by the air, the same condition will be found on the left after the transfer.

If obstruction exists in the ear itself, the hearing by the air will only return after the transfer to the degree which is to be expected with such obstruction. This was well illustrated in the case of Mlle. Card——, whose left tympanum was normal, but whose right indrawn and opaque, while the Eustachean tube on the right was impermeable. The transfer being made in her case, while the hearing by the bone is lost on the left, and that by the ear reduced to such a degree that the watch is only heard on contact, the hearing on the right is recovered for sounds conveyed by the bone, while that for sounds conveyed by the air only returned to a slight degree. That is to say, the watch, which before the transfer was only heard by the right ear, on contact was heard after the transfer at a distance of 10 centimeters. This recovery must be in her case considered a perfect one, as far as the auditory nerve is concerned. Both membranes being normal in any case, the same degree

of hearing is recovered by the previously anæsthetic ear, which existed before the transfer in the unaffected ear. This may be seen in the following experiment, the only one of which the details need be given, as it is typical of all, the variations between the different cases being such as are pointed out by Richer, and quoted above.

Experiment.—Mlle. Mig——. (Case IV. above.) Complete anæsthesia left, including the tympanum. Analgesia on the right. Loss of hearing by the bone on both sides; loss of hearing by the air on the left (does not hear the watch on contact); preservation of hearing by the air on the right to a high degree (hears the watch at 120 centimeters).

A large horse-shoe magnet was so placed that one of its poles was within a few centimeters of the left, or completely anæsthetic ear.

In a few minutes the sensation appeared in the left ear and the whole of the left half of the body. The watch, previously not heard on contact, was heard first on contact, then at a distance of a few centimeters, and at the end of seven minutes from the commencement of the experiment was heard at a distance of 115 centimeters. At this time a tuning-fork placed on the bone behind the left ear was not heard when the ear was closed. Touching the deep part of the ear was felt, but produced no unpleasant sensation. This ear, then, had now reached exactly the same condition of the right before the transfer.

At this time all sensitiveness to the touch had disappeared in the right ear, as well as over the whole right half of the body. The watch was not heard on contact, nor the tuning-fork against the bone. This ear had then lost its general and special sensitiveness, while the other had recovered exactly the amount which this had lost.

At the end of ten minutes from the commencement of the experiment the return oscillation commenced, and was completed in three minutes, at the end of which time the two sides stood in the same relation to each other as at the beginning. After an interval of three minutes the second oscillation commenced and lasted two minutes. The return commenced in three and a half minutes, and lasted a minute and a half.

Measurements taken during this return oscillation of the distances at which the watch was plainly heard, gave the following results:—

Time.	*Distance* (R.).	*Distance* (L.).
0	Not on contact.	115 mm.
30 sec.	6 mm.	40 mm.
60 sec.	58 mm.	Contact.
90 sec.	120 mm	Not on contact.

This table illustrates admirably the regularity with which the disappearance of function on one side is compensated for by its appearance on the other.

The third oscillation commenced in five minutes, and lasted only thirty seconds; the return commencing in four minutes, and lasting forty seconds.

The next four changes ranged from thirty to forty seconds, and the intervals were of about the same length. The character of the oscillations was always the same, and at the end of the fifth the experiment was discontinued.

The patient was found at the end of several hours in her original condition, completely anæsthetic on the left, and incompletely so on the right.

As it seems hardly necessary to give further details, I close with the following principal ·

Conclusions.—1. The sensibility of the deep parts of the ear, including the tympanum and middle ear, disappears in hysterical hemianæsthesia with that of other parts of the body, and in the same degree.

2. The degree of deafness corresponds with that of the general anæsthesia, being complete when the latter is complete, and incomplete when the latter is incomplete.

3. When the loss of hearing is incomplete, the deafness for sounds conveyed by the bone exceeds that for sounds conveyed by the air.

4. When the transfer is made, the hearing as well as the general sensibility of the deep parts of the ear improves on one side (allowance being made for accidental lesions in the ear itself) in exactly the same degree in which it disappears on the other.

ON THE SPASMODIC PARALYSES OF INFANCY.

BY JAMES ROSS, M.D., F.R.C.P.

(Continued from p. 363.)

In the first part of this paper, which appeared in the last number of 'BRAIN,' I described a case (Case II.) of double athetosis with aphasia. It may be worthy of notice in passing, that Sir Walter Scott's description of Nick Strumpfer, the dwarf in 'The Pirate,' corresponds so accurately to the phenomena presented by my patient, so far at least as the facial contortions and disorderly movements are concerned, as to leave little room for doubt that the picture was drawn from real life. The Udaller, with his two daughters, pays a visit to their kinswoman, Norna of the Fitful Head. After a vigorous knocking, "the door opened, and displayed, to the alarm of Brenda, and the surprise of Minna herself, a square-made dwarf, about four feet five inches high, with a head of most portentous size, and features correspondent—namely, a high mouth, a tremendous nose, with large black nostrils, which seemed to have slit upwards, blubber lips of an unconscionable size, and huge wall-eyes, with which he leered, sneered, grinned, and goggled on the Udaller as an old acquaintance, without uttering a single word." The Udaller facetiously assured his daughters that the dwarf could "keep his mistress's counsel, and never told one of her secrets in his life." "The ugly dwarf grinned ten times wider than before, and showed the meaning of the Udaller's jest by opening his immense jaws, and throwing back his head, so as to discover that in the immense cavity of his mouth there only remained the small shrivelled remnant of a tongue, capable perhaps of assisting him in swallowing his food, but unequal to the formation of articulate sounds.

Whether this organ had been curtailed by cruelty, or injured by disease, it was impossible to guess; but that *the unfortunate being had not been originally dumb, was evident from his retaining the sense of hearing.*" "Having made this horrible exhibition, he repaid the Udaller's mirth with a loud, horrid, discordant laugh, which had something in it more hideous, that his mirth seemed to be excited by his own misery." It may safely be assumed that the description which even such an acute observer as Scott gives of the small and shrivelled tongue, was biassed by the preconceived theory that the inability to articulate must have been occasioned by a local defect of the tongue. The last case which I reported in the first part of the paper was an example of congenital hemiplegia, with a defect in the parietal bone on the opposite side. The following case, kindly sent to me for examination by my friend Dr. Cullingworth, is an example of congenital hemiplegia, without any other defect being present.

Case VI.—Margaret H. K., aged 8 years, came under my observation on July 19, 1881. An aunt of the child, under whose care she is at present, stated that the patient never suffered from any illness, and is particularly positive that she never had convulsions. The parents are convinced that the left hand was paralysed at birth, and it was noticed that she never moved it in early infancy.

Present condition.—The subject is a little girl, 3 feet 11 inches in height, well-proportioned, healthy in appearance, of fair complexion, and with a bright and intelligent countenance. She goes to school, and has attained the second standard, and is supposed to be a very good scholar for her years. The left arm and leg are partially paralysed. The muscles about the left shoulder-joint are not implicated in the paralysis, but the patient cannot fully extend the fore-arm upon the arm, or supinate the fore-arm, and considerable resistance is offered to passive supination of it. All the movements of the affected hand and fingers can be performed, but they are not fully co-ordinated, and the patient experiences considerable difficulty in picking up a pin, or grasping small objects with this hand. The tendon reflexes are not decidedly increased. The various segments of the left lower extremity are extended upon one

another, the foot is kept in a state of extreme talipes equino-varus, and considerable muscular tension is excited by attempts at passive movements of the limbs. The patellar-tendon reflex is only feeble in the left, and absent in the right leg. Ankle-clonus cannot be elicited. The following measurements were taken :—

		Right.	Left.
Clavicles	Length	4 inches.	3¾ inches.
Upper arm	"	8¾ "	8½ "
"	Circumference	7 "	6½ "
Fore-arm	Length	6½ "	6 "
"	Circumference	6¾ "	6¼ "
Hand	Circumference	5½ "	5½ "
Thigh—From ant. sup. spine of ilium to external condyle		13½ "	12 "
"	Circumference, thickest	14 "	13 "
Legs, calves	Circumference	9¾ "	9 "
"	Length from head of fibula to external malleolus	10½ "	9½ "
Foot	Length	7½ "	7 "
"	Circumference of instep	7½ "	7½ "

The face is quite symmetrical, and there is not the slightest evidence that the facial muscles have been implicated. The faradic contractility of the affected muscles is normal, and there are no sensory disturbances. The skin over the left leg and foot is blue and mottled, and becomes much colder than that of the right leg after a short exposure.

The case which has just been described demands no lengthened comments. A porencephalous defect is probably present in the right hemisphere of the brain. This case is remarkable for the complete absence of idiocy or any mental defect. It will be interesting to know whether epilepsy will become established at puberty, as occurs in cases of unilateral atrophy of the brain.

My thanks are due to Dr. Cowan, of Newchurch, for sending me the following case, and for obtaining a post-mortem examination after the child had died from an attack of croup.

Elizabeth B., aged 2 years and 5 months, came under my observation as an out-patient at the Royal Infirmary, some time in November last year. The following notes of her case were taken on Dec. 19, 1881. The parents are healthy, and have several other children, all of whom are healthy. The mother has not had any miscarriages. The child appeared to have

been well-formed and healthy at birth, and it was only at three months of age that the parents observed that she could not hold her head up, and that her hands were stiff. The infant had not at any time suffered from convulsions, and has been singularly free from any infantile disease.

Present condition.—The child is small for her age, but fairly nourished. The lower extremities are slightly flexed at the hip and knee-joints, the feet are maintained in positions of talipes equinus; the muscles are rigid, and the spasm is much increased on any attempt being made to alter the position of the limbs by passive motion, the spasm being particularly manifest in the adductors of the thighs. The spasmodic rigidity of the muscles of the lower extremities also becomes much increased when an attempt is made to place the child erect on the floor. The muscles of the calves then contract powerfully, and the heels become elevated, so that only the points of the feet touch the ground. The adductors also contract strongly, so that the limbs are closely applied to one another when they are in the same plane, and become crossed when one is in front of the other. When the patellar tendon is struck, a spasm of all the muscles of the lower extremity supervenes, so that a true knee-jerk cannot be elicited. Ankle-clonus cannot be provoked in either foot. The muscles of the upper extremities are also in a state of spasmodic rigidity, and the various segments of the arms are held in more or less fixed positions. The upper extremities are held in symmetrical positions. The arms are abducted, so that each elbow is about four inches removed from the body; the fore-arms are semi-flexed on the arms, and pronated; the hands are slightly flexed in the fore-arms, and a little inclined to the ulnar borders; the thumbs are flexed into the palms; and the fingers are semi-flexed over the thumbs. It is impossible to produce complete extension at the elbow-joint by passive motion, and any attempt at altering the position of the limbs increases the spasmodic rigidity of the muscles. The patient can voluntarily grasp an object with either hand, but the movements made in order to reach the object are irregular and uncertain, and it is impossible for her to pick up small objects.

The head is habitually kept bent forwards, so that the chin rests on the sternum. The sterno-cleido-mastoid muscles are tense and prominent, the left muscle being more prominent than the right, and consequently the head has a lateral inclination towards the left. The child can raise her head by a voluntary effort, in order, for instance, to look at an object placed above the level of the axes of vision, but the necessary effort cannot be long sustained, and the head soon assumes its bent and inclined position. The child does not speak yet, but the parents state that she makes an effort to utter a few simple monosyllables, but she could not be persuaded to do so in my presence. I could gather from the description of the parents that such attempts are accompanied by contortions of the mouth and tongue, caused, doubtless, by spasmodic contractions of the muscles.

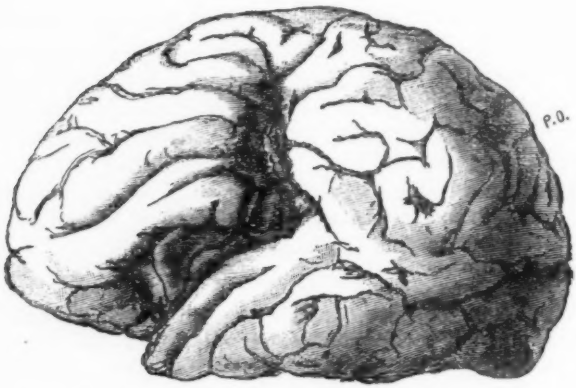
A few weeks after this report was made the child suffered from measles, and subsequently from an attack of croup, from which she died on Jan. 22, 1882.

The post-mortem examination was conducted on the evening of the 24th of January, by Dr. Cowan, Dr. Coutts, and myself.

The skull was symmetrical, smaller, if anything, than corresponds with the age, the transverse axis from one parietal bone to the other being unusually short. On the calvarium being removed, the dura mater appears in every respect healthy; but when it is reflected back, a deep sulcus is observed on each side, corresponding to the positions usually occupied by the fissures of Rolando. The external opening of these sulci is bridged over by the visceral layer of the arachnoid. Each sulcus extends from the point of bifurcation of the Sylvian fissure upwards to near the longitudinal fissure, a distance of 1½ inch. The lateral walls of each sulcus are formed by an infolding of the grey matter of the cortex, and these walls are so closely applied to each other that there is no great cavity left between them. In depth each sulcus extends the whole thickness of the centrum semi-ovale, and opens into the roof of the corresponding lateral ventricle. The opening into the ventricle on each side is only large enough to admit the little finger, and each opening is surrounded by a ring of grey matter, having all the naked-eye appearances of

the cortex. The cavities communicate freely with the ventricles without being covered by the ependyma. The ventricles are of normal size, or only slightly dilated, and only about one ounce of serous fluid has escaped during the removal of the brain. With regard to the fissures and convolutions, those in the neighbourhood of the defect are radially directed towards the centre of the cavity (Fig. 1). The ascending parietal and

FIG. 1.



ascending frontal convolutions appear to be absent on both sides. The posterior limb of the Sylvian fissure is very short, and runs almost vertically instead of horizontally as usual. The first and second temporo-sphenoidal convolutions have also a vertical inclination. The fissure of Rolando is replaced by the sulcus already described; the anterior wall of the sulcus is formed by the posterior extremities of the first and second frontal convolutions, and the posterior wall by the angular and supra-marginal gyri and, at the upper extremity, by the parietal lobule. On the internal surface of the hemispheres the calloso-marginal fissure does not extend backwards as far as usual, and the paracentral lobules are only imperfectly represented. The crura cerebri, pons, medulla oblongata and spinal cord appeared quite normal to the naked eye.

A microscopical examination of the grey matter forming the walls of the cavity show a structure more or less similar to

that of the healthy cortex. The giant cells of the inner division of the third layer of the cortex are, however, absent. This division contains large, round, nucleated cells, but they are entirely destitute of processes; and indeed absence of processes is a marked feature of all the cells observed, the cells being in this respect like those met with in the cortex of the brain in the embryo. The spinal cord on microscopical examination appeared in every respect normal, except that the lateral columns were somewhat smaller than the corresponding parts in a healthy cord. The anterior pyramids of the medulla oblongata are not much more than half the size of the pyramids of the medulla from a healthy child of the same age. The accompanying woodcut (Fig. 1) is from a sketch of the right hemisphere of the brain, kindly taken by Mr. A. H. Young.

Remarks.—In this case it would appear that the porencephalous defects were due to a simple arrest of development, and no cicatricial tissue was found in the walls of the cavities to indicate that they were caused by a destructive process. When the case came for the first time under my observation I was somewhat puzzled as to the nature of the lesion. As the mother was attended during her confinement by a midwife, it occurred to me that probably undue traction had been made on the head, thus injuring the upper cervical vertebræ, and giving rise to a pachymeningitis and subsequently to a transverse myelitis. But although such a course of events might account for the paralysis and contractures, it would not account for the affection of speech present. With regard to the affection of speech, it was difficult to determine whether it was of the nature of aphasia or of dysarthria. I felt inclined to believe that the disturbance of speech was of the latter character, inasmuch as the symptoms were almost in every respect similar to those observed in the case of James W. (Case IV.) when he first came under my care. It thus occurred to me that a single lesion in the pons might account for all the symptoms, for the same reasons which induced me to believe that such a lesion would account for the symptoms in the case of James W.

Against this view were to be set the facts that the child had a

very small head, and the expression of a microcephalic idiot. The condition which was actually found requires no further comment.

Spastic Paraplegia.—Let us now pass on to the consideration of the affection which may be termed *Spastic paraplegia of infancy.* The following four cases may be taken as good examples of this affection.

Case VII.—Wilfred R. L., aged 8 years, was kindly sent to me by Mr. Southam in April 1882. The parents of the child are living and healthy. The mother has had four children; one died in infancy from scarlet fever, and the two living, besides the present boy, are strong and healthy. No tendency to any kind of nervous disease can be discovered in the family. The mother thinks that the head of the child was born first, but she states that it was a "cross-birth," and that one foot was presenting at first. She does not know whether the operation of "turning" was undertaken by the medical man in attendance. The mother observed that the right ankle was slightly deformed when the child was a few days old, but nothing seriously wrong was discovered until it was time for it to walk. The patient never had convulsions.

Present condition.—The boy is well developed, with a ruddy complexion and healthy appearance. Height 41 inches. The right foot is in a permanent condition of talipes equino-varus; the toes are spread out, and there is a slight hyper-extension at the torso-phalangeal, and flexion at the phalangeal joints. The left foot is similarly deformed, but to a much less extent than the right. The boy can separate his thighs well, but to a less extent than is usual in health, and during the action the adductors can be felt in a state of spasm. The leg cannot be fully extended on the thighs, or the thighs on the trunk. The patient habitually stands on the sole of the left and the toe of the right foot, the latter being considerably in advance of the former. In this position the vertebral column is slightly curved to the right in the lumbar region, while there is a slight compensating curve to the left in the dorsal and lower cervical regions, and another to the right in the upper cervical region. The buttocks are somewhat projecting, and the trunk is slightly inclined forwards, so that a plumb line let fall from

the most prominent of the spinous processes of the vertebræ falls well within the sacrum. The dorsal curve which is so prominent a feature of pseudo-hypertrophic paralysis is absent. When the boy is made to stand with both feet in the same antero-posterior plane, he experiences great difficulty in bringing his left heel to the ground, and the muscles of the calf are then very tense and stretched, but he is wholly unable to bring his right heel lower than to within one inch from the ground.

Whenever he attempts to walk, both heels become immediately elevated, the left heel being one inch removed from the floor when the leg of the same side is active, and the right heel two and a half inches removed from it under similar circumstances. The boy consequently walks on the balls of his toes, and both feet are in positions of talipes equinus or equino-varus, the deformity on the right side during locomotion being so great that the foot assumes almost a vertical position. When the right leg is being moved forwards, the various segments of the extremity become nearly extended upon one another, with the exception of the slight degree of permanent flexion at the knee-joint. The distance between the trochanter and the toes is increased, and consequently the toes cannot clear the ground, and the leg cannot swing forwards by its own weight as in health. In order to effect the forward movement the boy leans his body well over to the left, thus elevating the right side of the pelvis along with the trochanter, and allowing the right leg to be projected forwards in one piece. But notwithstanding the elevation of the pelvis on the right side, the toes are only partially cleared off the ground, and every forward movement of the foot is attended by a scraping noise, and the toe of the boot on the right side is much worn. The forward movement of the leg is a compound one. The leg is moved forwards in one piece by the flexors of the thigh upon the body, but at the outset of the movement an outward inclination is given to the foot by a voluntary contraction of the abductors of the thigh, in order to raise the toes off the ground more effectually, while at the close of the movement the strong involuntary contractions of the adductors predominate, and the foot is dragged over to the left, and planted in front of the left foot. The foot, therefore,

instead of being moved forwards in a straight line, describes a semicircle, with its convexity to the right. When the right foot is brought to the ground and the right leg becomes active, the left leg is moved forwards in a similar manner to that described as occurring on the right side. But inasmuch as the toes are much less depressed on the left than the right side, the pelvis requires to be less elevated, and the toes do not drag on the ground with the same scraping noise. The alternate elevations of the pelvis on each side during the forward movements of the legs give to the gait a waddling appearance, not unlike that observed in pseudo-hypertrophic paralysis, but readily distinguished from it by the absence of the dorsal curve.

The patellar-tendon reflexes are very lively, probably exaggerated, but ankle-clonus cannot be elicited on either side. There is complete absence of all sensory disturbances, the electrical reactions of the nerves and muscles are normal, the nutrition of the muscles is preserved, and there are no disorders of the functions of the bladder or rectum.

The head is unsymmetrical, the parietal protuberance being well marked on the right side and deficient on the left. The circumference of the head is 20½ inches. The forehead is narrow, but otherwise well formed. The boy manifests a slight hesitation of speech, but there is no distinct stammering. The mother states that the boy's memory is very good, and that he is particularly quick at learning his lessons at school.

The following are brief notes of a similar case which came under my observation.

Case VIII.—T. M., aged 4 years, was brought to me by his parents in July 1881. The parents are healthy, and there is no evidence of a neurotic tendency, or predisposition to any constitutional disease in the families on either side. The mother had an easy confinement, and the head presented. The boy never suffered from convulsions in infancy, and he always enjoyed good health. Soon after birth it was noticed that the feet were deformed and stiff, but not much attention was paid to this condition until the child attempted to walk.

Present condition.—The patient is small for his age, but well developed, and of healthy and intelligent appearance.

On standing, he cannot bring both heels at once to the ground. Whenever he walks, a spasm of the muscles of the calf of the active leg supervenes, which elevates the heel to a distance of 1 to 1½ inch from the ground, so that the patient walks on his toes. The toes of the boots are worn, and he is very liable to catch the toe of the advancing foot against any slight inequality on the ground, and is consequently very liable to fall; but there is no scraping of the toes on a level floor. The gait is awkward and waddling, but there is no dorsal curve; the sacrum projects slightly behind the most prominent of the spinous processes of the vertebræ. The patellar-tendon reflexes are exaggerated, and a considerable degree of muscular tension is provoked by passive movement, but ankle-clonus cannot be elicited on either side. The electrical reactions of the nerves and muscles are normal, and there is no muscular atrophy. There are no sensory disturbances, and no disorder of the functions of the bladder and rectum.

The head is symmetrical, measures 19 inches in circumference, and no squinting or other disorder of the cranial nerves has been observed. The patient can speak fluently, and is believed by his parents to be as intelligent as other children of the same age.

Case IX.—James B., aged 4 years, was brought to the Royal Infirmary as an out-patient on August 16, 1882. The father and mother are both healthy, and no hereditary disease can be traced on either side. The parents have been married five years, and this boy is their only living child. Another boy died when six months of age, from convulsions. The mother had a tedious labour when confined of this child, but instruments were not used to facilitate delivery. When the child was born, his head was noticed to be distorted, the one side being much more prominent than the other. Immediately after birth the infant was seized with convulsions, and these continued to recur for the first five days after birth. He had no difficulty in swallowing after the convulsions ceased, and he does not appear to have dribbled more than usual. At six months of age he had two slight fits, but they appear to have passed off without leaving any trace of disease. He also suffered from some abscesses during childhood, but the slight

marks left by these show that they were only subcutaneous. With these exceptions the child has enjoyed good health, and it was not until he was two years of age that it was observed that anything was wrong with his legs.

Present condition.—The child is small for his years. He measures 33 inches in length, and 17 inches round his waist. His head is well formed, but small, measuring only 18 inches in circumference. The face is also small, and the boy has a sharp, restless, but fairly intelligent expression. When the child is sitting there is no perceptible deformity of the lower extremities; but when his feet are placed on the ground, the heels become immediately elevated, so that he stands on his toes. The thighs are slightly flexed on the body, and the legs on the thighs; the toes are somewhat inverted, and

FIG. 2.

the knees are strongly drawn together by a spasm of the adductors of the thighs. The boy is unable to stand or walk unsupported, and when he walks by the aid of a support, the knees rub against each other. When the patient

stands or walks, the muscles of the lower extremities, especially the gastrocnemii and the adductors of the thigh, become hard, prominent and tense (Fig. 2); the patellar-tendon reflexes are exaggerated, but ankle-clonus cannot be elicited. The child is able to speak without any stammering, but his speech is drawling, and more like that of a child of two than of a boy of four years of age. There are no sensory disturbances, and the bladder and rectum are unaffected. The accompanying woodcut is from a photograph, kindly taken for me by my friend Dr. Larmuth.

Case X.—Henry F., aged 8 years, entered the Manchester Royal Infirmary on August 1, 1882. His parents are quite healthy. This boy is one of a family of four children, all of whom are healthy, the other three being free from any defect whatever. The mother of the boy has had six miscarriages, some of them occurring at the third, and some at the sixth month. No other evidence of a syphilitic history can be ascertained. The boy was quite healthy as an infant, but at six months of age he began to have fits, and had as many as two or three in the course of a day, and the fingers and toes remained strongly flexed in the interval between the convulsions. It was observed that his head was large as an infant, and he was then supposed to have water in the head.

Present condition.—The boy is well-nourished and healthy-looking. His head is large, measuring 22 inches in circumference, 9 inches in the longitudinal and 6 inches in the transverse diameter; the forehead is projecting; the fontanelles are closed, but depressed; and the eyes are normal. When the boy is sitting, there is no manifest deformity of the lower extremities; but when he attempts to stand or walk, the heels become elevated, and he has to balance himself on his toes. The legs cannot be fully extended on the knees, and the knees are drawn together by a strong contraction of the adductors of the thighs. The thighs are also during station slightly flexed on the trunk, and the toes are inverted. The knees rub against each other when the patient walks. The patellar-tendon reflexes are exaggerated, and a slight ankle-clonus can be provoked on the left side, but not on the right. There are no sensory disturbances and no bladder troubles.

Although the hands were implicated in the spasm when the patient suffered from fits, they are now normal. The feet become dark red on exposure, and are very liable to suffer from cold. The faradic contractility of the muscles affected by the spasm is normal.

The four cases which have just been described, although differing widely from one another in many respects, are good examples of the spastic paraplegia of infancy. The occurrence of spastic paraplegia in infancy is, if I may judge from my own experience, by no means rare. Such cases have been long known to orthopædic surgeons. Even as far back as 1840,[1] Heine described three cases of spinal paralysis of infancy characterised by "a spastic peculiarity of the contracted muscles;" and the accompanying figures, as well as the description of these cases, leaves no room for doubt that they were examples of the disease under consideration at present. The clinical features of spastic paraplegia have also been faithfully described by Dr. Little in 1853,[2] and again in 1862,[3] and by Mr. Adams in 1866.[4]

Stromeyer also in 1864 directed attention to Dr. Little's observations, and described this affection under the name of "permanent tetanus of the extremities in children."[5] In 1877[6] Erb described three cases of this affection; a few months later[7] he reported two other cases; and in 1877, and again in 1880, Dr. Gee communicated several cases of the disease.[8] When Erb assimilated these cases clinically to the spastic paraplegia of adults, he suggested to the mind that the former

[1] 'Beobachtung über Lähmungszustände der untern Extremitäten,' von J. Heine, 1843, p. 61 et seq.

[2] 'Deformities of the Human Frame,' by W. J. Little, M.D. Lond. 1853.

[3] "On the influence of Abnormal Parturition, Difficult Labours, Premature Birth, and Asphyxia Nonatorum, on the mental and physical condition of the Child, especially in relation to Deformities," by W. J. Little, M.D. 'Obstetrical Transactions,' vol. iii. 1862, p. 293.

[4] 'On Club Foot,' by W. Adams. Lond. 1866. P. 46.

[5] Stromeyer's 'Handbuch der Chirurgie.' 2 Bd. 1864. Pp. 17 and 174.

[6] "Ueber die Spastische Spinalparalysie" (Tabes dorsalis spasmodique, Charcot). Virchow's 'Archiv,' Bd. lxx. 1877, pp. 241 and 293.

[7] 'Ueber das Vorkommen der "Spastischen Spinallähmung" bei Kleinen Kindern, Memorabilien aus der Praxis.' Heilbronn, 1877, 12 Heft.

[8] 'St. Bartholomew's Hospital Reports,' vol. xiii. 1877, p. 71, and vol. xvi. 1880, p. 97.

disease is, like the latter, due to a primary sclerosis of the lateral columns of the cord. He is, however, careful not to commit himself to this view, and he has even suggested that the infantile disease may be due to an arrest of development of some portions of the spinal cord, or at least of the nervous system.

But it becomes a question whether the disease is of spinal or of cerebral origin. The paraplegic distribution of the spasm, and the frequent absence of any distinctively cerebral symptom, would seem to indicate that the affection is, sometimes at least, caused by a spinal lesion. But on the supposition that this disease is a spinal one, the questions arise, whether it is due to a congenital absence of some of the fibres of the pyramidal tracts or to sclerosis of them? and, whether the paralysis becomes established before, at, or after, birth? Satisfactory answers to these questions can only be obtained by careful clinical reports with subsequent dissections. It is rather remarkable that in several cases which came under my observation, but of which I have not kept notes, the children were born with the feet first. It has occurred to me that injury might have been done in these cases to the vertebral column and spinal cord by undue traction during delivery. Such an injury might produce the disease in several ways. Injury to one of the vertebræ, for instance, might set up a limited pachymeningitis, with subsequent transverse myelitis, and the latter would cause a descending sclerosis of the pyramidal tracts. Compression of the spinal cord by the formation of a blood-clot between the injured vertebra and the dura mater might lead to a similar result. Transverse myelitis would affect the sensory as well as the motor mechanism of the cord, but it is well known that in the adult, sensory disturbances disappear much more readily than motor disorders; and this would be much more likely to take place in the infant, inasmuch as the pyramidal tracts are only imperfectly developed at birth. That the vertebral column may suffer injury during the extraction of the child at birth has been proved by dissections. A portion of a vertebral column from a child extracted by the feet was exhibited in 1876 to the Obstetrical Society of Leipzig,

by Ahlfeld,[1] showing that the bodies of two vertebræ were divided. The child lived nine days, and the existence of the lesion was not suspected before death. Out of 64 cases of extraction by the feet observed by Ruge, rupture of the vertebral column occurred 8 times. The rupture occurred in the body of a vertebra in the upper dorsal or cervical region, and in the line of the epiphyses; the anterior common ligament was torn, and the spinal cord was always found surrounded by an extravasation of blood at the seat of the injury. It is very probable that no child will long survive injuries so extensive as were found in the cases reported by Ahlfeld and Ruge, but it is quite probable that a slighter degree of injury may be quite compatible with life, or may indeed give rise to no very manifest symptoms.

But it is probable that hæmorrhage of the spinal membranes might give rise to a similar result in the absence of any rupture or other injury of the vertebræ. Out of 161 post-mortem inspections by Weber, of newly-born children from the 'Clinic' of Litzmann,[2] the vertebral canal was opened 81 times, and of these, extravasation of blood was observed 33 times, the hæmorrhage being very extensive in 19 cases. Of the 33 cases in which extravasation had occurred, the blood was found on the external surface of the dura mater alone, 23 times; both on the external surface of the dura mater and the arachnoidal sac, 4 times; in the arachnoidal sac and sub-arachnoid space, once; in the arachnoidal sac alone, 4 times; and in the sub-arachnoidal space once. Extravasation of blood of meningeal origin was also found within the cranium in these cases in addition to that in the spinal canal. A wound of the vertebral canal was not observed in any of these cases; it is therefore manifest that spinal meningeal hæmorrhage is by no means an uncommon occurrence at birth, and it is not to be supposed that all the children must necessarily die of a lesion which does not appear to be a very fatal one in the adult. It is therefore not improbable that some cases of spastic paraplegia may owe

[1] See Litzmann, 'Archiv für Gynäkologie.' Bd. xvi. 1880, p. 97.

[2] "Ein Beitrag zur Kenntniss der Spinaler Lähmung bei Neugeborenen," von C. C. M. Litzmann. 'Archiv für Gynäkologie,' Bd. vi. Berlin, 1880. S. 99.

their origin to a spinal meningeal hæmorrhage. Hæmorrhage into the substance of the spinal cord itself would injure the grey matter, and lead to atrophic paralysis of some groups of muscles, but no atrophy is ever present in the cases under consideration at present.

But it is possible that the spinal cord itself may suffer direct injury during efforts at extraction of the child. Parrot[1] records a case in which a midwife heard, while strongly pulling the feet of a child during birth, a crack which appeared to come from the upper part of the child's body. Death occurred on the sixth day after birth, and at the autopsy the spinal cord was found completely torn on a level with the sixth and seventh cervical vertebræ, a rent being also observed in the membranes on the same level. A clot of blood, which filled the whole breadth of the spinal canal, lay between the divided ends of the cord. It is not very clear whether the vertebral column was injured or not, but it would be difficult to imagine that complete rupture of the cord could occur without simultaneous rupture of one of the vertebræ.

But if it be possible for complete rupture of the spinal cord to take place under any circumstances, surely it is possible for partial rupture to take place. A slight degree of traction upon the spinal cord might injure some of the fibres of the pyramidal tracts, a considerable proportion of these being non-medullated at birth, while leaving the earlier developed fibres of the cord, as well as the grey substance, intact. The peripheral portion of the injured fibres would subsequently undergo descending sclerosis, and thus give rise to the spastic paralysis.

But although some cases of spastic paralysis may be accounted for by injury done to the vertebral column, spinal membranes, or spinal cord itself, during the act of birth, yet all cases cannot be explained in this manner. In many cases of spastic paraplegia the mothers have had a perfectly natural labour, and there can be little room for doubting that the spasmodic condition is fairly established at birth. In these cases we must assume that the lesion is a congenital one, and we

[1] 'Union Médicale,' 1870, Jan. 27, quoted by Leyden, 'Klinik der Rückenmarkskrankheiten.' Bd. ii. 1875, p. 88.

turn for an explanation of such cases to Erb's supposition that the spasmodic condition is due to an arrest of development of certain portions of the nervous system. Now the only portions of the spinal cord, arrested development of which would cause spastic paraplegia, are the pyramidal tracts, and arrest of development of these tracts is well known to occur sometimes. These tracts have been found absent by Flechsig in the spinal cords of anencephalous children; they were imperfectly developed in a case of porencephalus reported by Kundrat already alluded to, and also in the case of porencephalus just described by myself. It would appear, however, that arrest of development of the pyramidal tract is associated in the cases hitherto examined with a congenital lesion in the motor area of the brain, and it will probably be found that these conditions always co-exist. If that be the case, we may expect that at least a considerable proportion of cases of spastic paraplegia are really cases of bilateral hemiplegia, caused by a porencephalous defect of the cortical motor centres of the affected limbs, and arrest of development of the fibres of the pyramidal tract, which would have sprung from these centres. Even a clinical consideration of cases of spastic paraplegia suggests that the brain is implicated in the disease.

Speaking of the more aggravated cases, in which the upper as well as the lower extremities are implicated, Mr. Adams says: "Frequently strabismus, and occasionally wry-neck, coexist with these deformities."[1] "The head," he continues, "frequently exhibits a close approximation in form to that commonly existing in idiots; and imperfect development of the intellectual faculties sometimes accompanies this sad condition. These children have not infrequently a vacant or half-silly expression of countenance, and yet are more intelligent than might be supposed. Their speech is often difficult and imperfect, and in consequence of this, the intellectual faculties are often supposed to be more impaired than they really are."

And even in the minor grades of the affection, when the spasm is restricted to the lower extremities, evidence is not

[1] 'Club-Foot: its Causes, Pathology, and Treatment,' by William Adams, F.R.C.S. 2nd Edition. Lond. 1873, p. 65.

wanting to show that the primary lesion is situated in the brain. In the case of James B. (Case IX.), for instance, the patient may be regarded as a microcephalic idiot; while Henry N. (Case X.), although not now suffering from any cerebral symptoms, had "fits" at six months of age; the fingers as well as the toes were contracted in the intervals between the seizures; and from the size and form of his head he was supposed to be affected with hydrocephalus. Even Wilfred R. L. (Case VIII.), who is a very bright intelligent boy, has an impediment in his speech, and his head is unsymmetrical; while T. M. (Case IX.) has a small head, although it is symmetrical.

The opinion that a large number, if not all cases of the spastic paraplegia of infancy are caused by a porencephalous defect of the cortical motor centres, along with an arrest of development of the corresponding parts of the pyramidal tracts, is here advanced with due reserve. The obtainable evidence only warrants us in regarding such an opinion as more or less conjectural, and we must be content to wait until careful dissections shall enable us to place the pathology of this affection upon a secure basis.

THE PHENOMENA OF ANGINA PECTORIS, AND THEIR BEARING UPON THE THEORY OF COUNTER-IRRITATION.

BY W. ALLEN STURGE, M.D.

In the following paper I shall endeavour to make an analysis of certain of the phenomena met with in cases of angina pectoris, with a view not only to throw some light if possible upon the explanation of angina, but also to carry the process somewhat further, and through it to seek for a solution to certain other problems in nerve physiology.

I will take as my text the description of his attacks given me by a patient who suffered from very typical attacks of angina. This patient, who had no perceptible organic heart-disease, described his attacks as consisting of a sudden severe gnawing pain at the apex of the heart, shooting through to the bladebone and down the left arm to the tips of the fingers, where he felt a strong tingling sensation. The pains were accompanied by palpitations of the heart, and towards the end of the attack by the belching of a quantity of wind. After the attack, there was soreness and tenderness of the skin of the left side corresponding to the seat of the pain. The attacks lasted from five to fifteen minutes. Let us endeavour to ascertain the meaning of this series of phenomena.

In the first place, what in physiological language is the explanation of a sudden severe attack of pain? It means that the person is conscious of an abnormal commotion in the grey matter of the sensory region of the brain, corresponding to the region of the body where the pain is felt. This commotion may have arisen spontaneously in this part of the brain, as is probably the case in most cases of epilepsy associated with sensory phenomena; or it may be due to the transmission

upwards of the results of a commotion in the grey matter of one of the subordinate parts of the nervous system, as for instance in the sensory grey matter of the spinal cord, or, lower still, in the grey matter of the sympathetic ganglia. With these, again, it is also possible that the commotion began spontaneously; or, on the other hand, they in their turn may only be passing onwards a commotion which has begun still further away from the centre, viz. (1) in the trunks of the sensory nerves, or (2), far more frequently at their peripheral extremities, in both of which situations the commotion is rarely spontaneous, but is almost invariably due to something outside the nerve or its extremity, which has brought the nerve into action.

Thus, then, an attack of pain localising itself in the heart may be due in theory to any one of the following causes:—

(1.) Some structural change in the heart substance, or some change in the heart contents, acting upon the peripheral terminations of the cardiac nerves.

(2.) A commotion arising spontaneously in the grey nervous ganglia of the cardiac plexuses of the sympathetic, the commotion being transmitted upwards to the spinal cord and brain.

(3.) A commotion arising spontaneously in the cervical ganglia of the sympathetic which give off branches to the cardiac plexus; or in the ganglia of the pneumogastric.

(4.) A spontaneous commotion in those portions of the grey matter of the spinal cord which are connected with these ganglia by the bands of communication passing from the cord to the sympathetic.

(5.) A spontaneous commotion in that part of the grey matter of the brain which in any of the previous cases would be the recipient of the nervous impulses passed up from below.

These, then, are the various conditions upon which the pain may in theory depend It would be interesting to inquire whether all of them are in practice met with. This, however, would require a long and difficult examination of the evidence derived from a large number of carefully observed cases. I am not sure whether in the present state of our knowledge

such an inquiry is likely to lead to satisfactory results. At any rate, for my present purpose it will be enough to say that in most cases of angina associated with structural disease of the heart or large vessels the primary irritation probably takes place at the peripheral extremities of the cardiac nerves; but that there is some evidence to show that there are varieties of angina beginning spontaneously in the nerve-centres. I shall say little, moreover, as to the *nature* of the commotion set up in the grey matter, beyond stating that it is probably closely allied to that met with in cases of epilepsy, and still more so to that present in the so-called epileptiform neuralgia. The relationship between epilepsy and angina pectoris was long ago pointed out and insisted upon by Trousseau.

Let us pass on to consider another feature of the attack: the pain having begun suddenly in the neighbourhood of the apex of the heart, shot through to the left bladebone, to the left shoulder, and down the left arm to the tips of the fingers.

The radiation of the pain from the limited district in which it began until it extended over a wide area is an evidence of an extension of commotion from one small patch of grey nerve-substance to other parts of grey matter more or less intimately associated with it. An extension of nerve commotion of this kind is a very familiar phenomenon to those who have carefully studied the phenomena of epilepsy. When an epileptic fit instead of destroying consciousness at once begins slowly with the production of an elaborate warning, it is no rare thing to find that a nerve commotion giving rise (say) to a tingling in one finger, gradually spreads until the tingling passes to the other fingers, and thence up the arm to the trunk; thence it may pass up the neck to the tongue, face and head, or downwards to the thigh, leg and foot; bright sparks may perhaps next be seen, or a noise heard in the ears. Then a few muscles may begin to twitch in the hand; the arm may next be drawn up, and at this point perhaps the patient loses consciousness, the commotion extending rapidly until the whole body is convulsed. These various sensations and phenomena following in regular succession do but mark

the extension of the original commotion over a wider and ever more widely extending area of grey nerve substance. This extension depends upon the intimate association of one nerve-cell with others, either in its immediate neighbourhood, or closely connected with it in function, an association established by means of commissural interpolar fibres passing from cell to cell. When one of these cells becomes abnormally excited, as in the case of a slowly beginning fit, the commotion makes itself rapidly felt in the other cells connected with it. These will tend in consequence to become similarly excited; and they, in their turn, will excite others still further removed from the first cell, and so on in endless progression.

So it is in angina pectoris. Whatever may have been the original cause of the commotion, the excitement having once been started in some one of the situations above mentioned, will tend to overflow from the original focus into neighbouring parts. But now comes the question, Why does the excitement adopt the peculiar mode of extension seen in the case of angina pectoris I have quoted, and which, as all will allow who have seen much of the disease, is a very common form of attack? Why, in other words, does the pain extend from the heart region down the arms, and down the left arm rather than the right? What possible nervous communication can there be between the heart and the arms, since the heart is supplied by the pneumogastric and sympathetic nerves, and the arms by the lower cervical spinal nerves?

It is at once evident that no intercommunication between these two regions can take place through the cardiac ganglia, nor through the ganglia of the sympathetic giving origin to the nerves which pass to the cardiac ganglia. Neither do the pneumogastric nerves or their nuclei of origin offer a means of intercommunication. Does the spinal cord offer a more probable solution to the problem? The cardiac nerves of the sympathetic come from the three cervical ganglia on both sides. Of these, the largest nerves are the two nerves which come from the middle cervical ganglia. The strands passing from these ganglia to the spinal cord pass in the trunk of the fifth and sixth cervical nerves; those passing from the inferior cervical ganglia, in the trunks of the seventh and eighth

cervical nerves. It is these four nerves which, in conjunction with the first dorsal, form the brachial plexus. We thus see that the region of the spinal cord which gives origin to the brachial plexus gives origin also to the greater part of the fibres which eventually find their way to the heart. Wherever, then, the original commotion may have taken place in an attack of angina pectoris; whether in the cardiac ganglia, or in the cervical ganglia of the sympathetic, or in the spinal cord; and whether the commotion be due to some peripheral irritation from disease of the heart's substance, or be a spontaneous outburst on the part of the nerve-cells implicated, it is evident that it is only when the commotion has begun in the cord, or has passed up to the grey matter of the spinal cord from the sympathetic, that any great extension in its area can take place such as that of which I am speaking. The next question is: Since the cardiac nerves come from the sympathetic on both sides of the body, and must consequently be connected with both sides of the spinal cord in an equal or nearly equal degree, how is it that in the vast majority of cases of angina pectoris, as in the case mentioned above, it is the left arm alone that is affected, and that affection of the right arm alone is so exceedingly rare, although in some few cases both arms may be involved? The answer to this question is of very great interest, for on it, I believe, depends the explanation of the theory of counter-irritation.

It is well known that in the grey matter of the spinal cord, in addition to the fibres passing from the nerve-cells to form the roots of the spinal nerves, and to those which pass upwards to the brain, there is an extensive system of commissural fibres connecting the two sides of the cord, and connecting also cells and groups of cells together. It is, indeed, upon this intercommunication of groups of cells that reflex action depends. It is no less certain that cells endowed with a similar function, as, for instance, that of motion, but connected with different regions of the body, are more or less intimately associated together. It might at first sight be thought that this grouping of cells of similar function would coincide strictly with the grouping of the fibres emanating from these cells to form nerve trunks, e.g. that the cells whose emerging fibres go to

form the motor part of the musculo-spiral nerve in the arm would form one group; and that those whose fibres go to form the motor part of the musculo-cutaneous nerve would form another. Similarly it might be expected that the cells governing the sensation of those parts of the skin of the arm supplied by the musculo-spiral nerve would be associated together; and that the same would be true of those governing the sensation in the skin supplied by the musculo-cutaneous nerve.

Now it has been shown by Remak, Erb, Ferrier, and others that this is not the case with the *motor* nerves, but, on the contrary, that the grouping of motor cells in the spinal cord depends on the physiological association of muscles in the production of certain movements, and not on the mere anatomical fact of their being supplied by the same nerve-trunk. In a paper I read before the Medical Society of London,[1] I narrated two cases proving the truth of this proposition most conclusively. The patients were suffering from progressive muscular atrophy, involving the arms. In one of them the only muscles involved were the biceps, the brachialis anticus, and the supinator longus—the three muscles, in fact, which are associated together in bending the elbow. In the other case, all the muscles of the arm and hand were completely atrophied *except* these three muscles. Now two of these muscles are supplied by the musculo-cutaneous nerve, and the other by the musculo-spiral.

If this is so with the *motor* functions of the cord, we might expect to find somewhat the same variety of association in connection with the *sensory* functions. In this case we should expect that the cells connected with the sensation of the upper arm, taken as a whole, would be associated in a group together, in spite of the variety of nerves distributed to the skin of this part; whilst, on the contrary, the cells governing the sensation of a scattered district supplied by one nerve, such, for instance, as that supplied by the musculo-spiral nerve, would belong to several groups, rather than be collected into one group. In other words, cells governing the sensation of neighbouring parts of skin would be more or less closely

[1] 'Proceedings of Medical Society,' vol. v.

connected with one another; whilst those supplying patches of skin at a distance from one another would be correspondingly separated.

But if this be true of neighbouring parts on the *surface*, we may expect that it will be equally true of parts which, like the skin and the structures lying *beneath* it, are contiguous to one another; thus we should expect to find that the centres controlling the sensation of any particular portion of skin would be intimately associated with those governing the sensation of the subcutaneous fat, of the muscles, and even possibly of the bones, lying beneath that area of skin. Similarly, that the sensory centres for the skin over any particular organ would be associated more or less closely with those controlling the sensory functions of the subjacent organ.

This theory of association of cells in the spinal cord, according to physiological requirement and correlation, has, as I have already said, been proved to be true in the case of the motor functions, and I believe that the distribution of the pain in angina pectoris proves its truth in connection with the sensory functions.

Let us for the moment grant that it *is* true, and let us inquire what method of distribution of pain we might expect to find in connection with an attack starting in the nervous apparatus of the heart or great vessels, and spreading.

It would first attack the part, superficial to the heart, or great vessels; that is to say, the sternum and ribs and their muscles, and the skin covering them. Having reached the skin, it would tend to spread from one part to another, but this spreading would be more likely to follow certain directions than certain others; for instance, as we have followed the majority of the sympathetic cardiac nerves into the cervical enlargement of the spinal cord, we may expect to find that the pain will be confined to the upper extremities, rather than that it would pass from the skin of the thorax to that of the abdomen. Again, the skin of the left side of the thorax would be much more affected than the right side, because of the situation of the heart to the left, and therefore as the extension of the pain goes on, it will be the left arm that will be chiefly involved. At the same time we should expect that in

severe cases the radiations would cross to the right side, and that the right arm might be affected, though rarely, if ever, to such an extent as the left. We should also expect that it would be exceedingly rare to find that the pain passed to the right arm without affecting the left.

But the original commotion in the cardiac centres will not only affect those parts of the nerve-centres governing the sensations of the front part of the heart or its large vessels, but all parts will partake in it. Consequently the radiation to contiguous districts will take place not only towards the front, but in other directions also. Hence we may expect to find that the patient will complain of pain in the left scapular region, whence also the way is not far to the left arm. The radiation downwards would tend to pass to the stomach, and we might thus look for some affection of that organ. We should be prepared to find also that there would be more or less affection of the lungs.

Let me narrate very briefly the account given of their attacks by a number of different patients. It will be remembered that the patient who has served as the text for this paper experienced a severe gnawing pain near the heart's apex, which extended over the whole cardiac region, shot through to the left bladebone, and down the left arm to the fingers. At the end of the attack he belched a quantity of wind. The attack left a soreness and tenderness in the skin of the left side of the chest. Another patient of mine, suffering from severe aortic valvular disease, had attacks in which he felt a sudden pain beginning over the heart, going back to the left shoulder-blade, and down the left arm to the finger tips, where he had a strong sense of tingling. Sometimes the pain extended also to the right shoulder and down the right arm, but here it never extended farther than the elbow. At the end of the attack, he belched a quantity of wind for a few minutes. Occasionally he suffered from shortness of breath during the attacks.

Another of my patients suffering from aortic valvular disease had attacks which began with a sudden choking sensation at the top of the sternum, with intense dyspnœa; this was followed by intensely sharp pain over the apex of the

heart, which extended to the left shoulder and down the left arm, which felt numb and heavy. After a few minutes he began to belch a large quantity of wind, the eructation continuing for some time.

A fourth patient of mine, a woman without evident organic heart-disease, had attacks, almost always at night, in which she had sudden severe pain in the cardiac region of a shooting or dragging kind, which went down both arms, but down the left more than the right. The hands tingled, and she felt as though the nails were being dragged out. Towards the end of the attacks she belched a quantity of wind. After the attacks the arms felt numb and weak.

Trousseau describes the following cases:[1]—

(1.) A woman with aneurism of the aorta had attacks of sharp sudden pain in the precordial region, radiating to the base of the chest, where it produced an intense sensation of constriction; thence to the loins, up to the neck, down the left arm to the extremities of the fingers, leaving the arm numb and heavy.

(2.) Another woman, without organic heart-disease, had pain behind the sternum, passing to the left shoulder and down the left arm, causing numbness.

(3.) A man without organic disease had pain behind the lower part of the sternum, passing to the base of the neck and down both arms equally, giving rise to painful numbness.

(4.) A man with aneurism of the aorta had attacks beginning with a sense of suffocation, followed by severe pains behind the sternum, passing to the left shoulder and down the left arm.

(5.) A man without organic disease had attacks of pain behind the sternum, accompanied by intense dyspnœa. The pain passed to both arms, but chiefly to the left, giving rise to numbness. The attack ended with a strong desire to pass water, and with a sense of congestion in the nasal mucous membrane.

In the majority of these cases, then, we see that the attack, beginning in the cardiac region, passed to the left arm alone; in three it passed to both arms; but in only one was the right arm affected equally with the left. In no case was the right

[1] 'Clinique Médicale,' 4ème édition, vol. ii. p. 527 *et seq.*

arm alone affected. Trousseau mentions a remarkable case in which the right arm was affected and not the left; but in this case the pain was entirely confined to the *right* side of the chest, and hence can scarcely be looked upon as one of ordinary angina pectoris, the case being probably one of epileptiform neuralgia of certain intercostal nerves of that side.

What evidence is there to show that the pain in the cardiac region was situated in the skin, covering that region as well as in the heart or its dependencies? An answer to this question is of importance, if my theory be correct that the pain passes from the deep organ to the superficial structures over it, and thence spreads along the skin to the arm, &c.

In the first place, I would quote the statement of my first-mentioned patient, who said that the skin over the heart was sore and tender to the touch after the attacks. Does not this recall at once the tenderness over a neuralgic district; and, on the other hand, if the intercostal nerves had been unaffected, why should the skin be tender? Again, Trousseau describes the case of a man with an aortic aneurism subject to attacks of angina pectoris, who after his attacks had *numbness of those parts of the chest where he had experienced the pain.*

In the next place, I would quote the description so often given by patients, and upon which Trousseau and other authors lay so much stress, viz. that even where there is no actual dyspnœa, the patient ceases at the height of the attack to draw breath, or draws it as lightly as possible, on account of the pain that it gives him to do so. Does this description not resemble exactly the condition of a patient with severe intercostal neuralgia—severe pleurodynia?

It will be seen that in several cases the patients made special mention of pain in the bladebone following on that in the precordial region. Walshe speaks of "pain shooting to the mid-dorsal spine" as not infrequent. This would appear, therefore, to be a radiation independent of that over the præcordium, and if this be so we should be justified in referring it to an extension from that portion of the cardiac centres of the spinal cord governing sensation in the back of the heart.

In all my cases gaseous eructation from the stomach

was a marked feature, and most authors who have treated of angina mention it as a frequent symptom. This is the most usual form of implication of the stomach, and it is one of very great interest, as proving conclusively the influence of the nerves of the stomach over the secretion of gas in that organ. Just as the connection between the heart and its superficial neighbours is evidently established through the sympathetic nervès passing to the cervical spinal cord, so here the connection between the heart and its deep neighbour the stomach is probably made through the pneumogastric nerves. If therefore there be a form of angina having its origin in the cardiac ganglia, and another form having its origin in the centre in the spinal cord controlling the heart, as is by no means improbable, we should expect that the former variety would be accompanied by stomach disturbance, and that the latter would not. The materials at my disposal are not sufficient to enable me to treat further of this point; but it will be an interesting question to ascertain whether the anomalous cases of angina not connected with organic cardiac disease, and not following the more usual methods of distribution (some of which cases may be supposed to arise in the spinal centres), present the symptom of eructation as frequently as the more typical cases of angina, or those definitely connected with heart-disease. This symptom of gas secretion under purely nervous influence is a most curious one, and worthy of further examination. I do not propose, however, to go further into this branch of the subject on the present occasion, beyond calling attention to the probable connection between the gaseous eructation of angina pectoris and the remarkable eructations of gas from the stomach in certain hysterical patients; the well-known phenomena of phantom tumour; the eructations which follow the fit in some cases of epilepsy; and probably also certain forms of flatulent dyspepsia in neurotic subjects.

As regards radiation of the nerve-commotion from the cardiac nerve-centres to those of the lungs, this is less frequently seen. In one of my cases dyspnœa was a prominent feature, and it was so, moreover, in two of the cases narrated by Trousseau. Walshe says that real dyspnœa is exceedingly

rare in angina pectoris; but in my own case above mentioned, there could be no doubt of the presence of real and most urgent dyspnœa.

The infrequency of lung symptoms may be taken, I think, as an indication that the affection of the stomach in angina pectoris is due to the close connection of the cerebro-spinal centres controlling the heart with those controlling the stomach, and that this connection depends upon the close proximity of the organs in the body, rather than upon the fact that both organs are supplied by the same nerves. Were it true that the almost constant association of symptoms in the heart and stomach in angina pectoris is due to the latter cause, it would be difficult to explain why the lungs, which also derive a large part of their nerve supply from the same nerves, are so very much less frequently affected. If, however, the explanation I have offered be the true one, then we should expect that, as by far the larger part of the lungs is situated at a distance from the heart, the communication between the heart and lung centres in the cerebro-spinal axis would not be particularly close, and that the lungs would only be affected in special cases.

In some of the more severe cases the radiation is much more extensive than to the back, left arm, stomach or lungs. We have already seen that it may go to the right arm; but it may also go up to the head down the trunk, and even to the lower limbs. In one of Trousseau's cases above described it affected the bladder and the nasal mucous membrane.

One of the most instructive phenomena of radiation to my mind is the sense of intense constriction, which is occasionally complained of. One of Trousseau's patients described it as being like a bar of iron violently tightened round the chest. Does this not at once recall to our minds the *douleur-en-ceinture* of spinal-cord disease which we have every reason to believe to be due to the involvement of the sensory centres of the intercostal or lumbo-cutaneous nerves? Its presence in angina pectoris is a fresh proof, if such be required, that wherever in angina pectoris the original irritation may have been, the seat of the commotion of which the patient is conscious is in the spinal cord. It shows, moreover, the inti-

mate connection between the spinal centres of deep organs, and those for the superficial structures covering them; for there is much less evident nervous connection between the cardiac ganglia and the intercostal nerves, than between these ganglia and the arms; yet we see that a commotion in the spinal cardiac centres may propagate itself rapidly to the centres from which emanate the sensory fibres of the intercostal nerves.

And now for the bearing of these remarks upon the theory of counter-irritation. When we use the actual cautery to the skin over the spines of the vertebræ in a case of disease of the spinal cord, what are we doing? We are producing a profound impression upon the peripheral endings of the nerves of the skin over the diseased part. We produce, moreover, a moderate degree of inflammation in the skin and parts immediately subjacent to it; but considering the depth at which the diseased part lies below the surface, we cannot imagine that the direct effect of the burn reaches so far inwards. Neither is it possible to suppose that the small amount of blood drawn to the skin by this slight degree of circumscribed inflammation can modify in any perceptible degree the amount of blood in the deeply subjacent organ. Can we not from our knowledge of what takes place in angina pectoris deduce an explanation of the effect of counter-irritation? An analysis of the phenomena of angina pectoris showed the intimate relationship which exists between the nerve-centres governing adjacent structures. We saw there how an irritation at the periphery of the nerves governing the deeply-seated organ, an irritation arising from heart-disease, aneurism of the aorta, &c., caused a commotion in the spinal centres with which those nerves are connected; that this commotion rapidly passed to centres from which emanate the nerves supplying the parts superficial to that organ, and that in these centres also a violent storm arose.

Can we not easily imagine the reverse; that an irritation of the peripheral endings of the nerves supplying the superficial structure, should set up a commotion in the spinal centres to which these nerves pass; that this commotion should extend to the closely-related centres governing the

subjacent organ or structure, and should profoundly affect this structure?

As a matter of strict fact this reverse process does sometimes take place in connection with the heart. Let us refer once more to Trousseau's masterly lecture on angina pectoris. One of his patients without organic heart-disease, but with a gouty history, had attacks *beginning in the left arm* and passing upwards to the chest, where the pain was chiefly confined to the cardiac region; here the pain was so intense that it seemed to the patient himself as though he must die. The attacks lasted three minutes, and in all points resembled attacks of angina pectoris, with the exception that the radiation of the pain took place in an inverse order. Another patient, without organic heart-disease, but who was also gouty, had attacks beginning in the muscles of the left arm, and thence passing to the cardiac region. In a third case the pain began in the two shoulders, and rapidly passed to the neck, to the tongue, to the arm, and to the chest. Here also there was gout, but no organic heart-disease.

In a fourth case the pain began in one of the dorsal vertebræ. It passed thence to the left arm, and finally to the cardiac region. No organic heart-disease was discovered in this case.

These cases, it is true, were not traceable to any morbid condition of the peripheral ends of the nerves of the arm, the primary commotion probably taking place in the spinal centres governing the sensation of the arm; but they serve to show how readily a commotion in these centres passes to the centres governing the cardiac region; and if a commotion beginning spontaneously in the spinal centres for the arm can thus so readily be transmitted, a similar commotion of these centres, induced by peripheral irritation, would do so with equal ease.

But if I am correct in believing that the evidence derived from an analysis of the phenomena of angina pectoris and allied conditions points to an intimate communion in the spinal cord between the sensory centres for superficial parts, and those for subjacent organs or other structures, ought we

not to find corroborative evidence of this fact in other physiological or pathological conditions?

It is less easy than might at first sight appear to get such evidence; since severe superficial localised lesions, not in themselves affecting deeper structures, are not very common. The following conditions must not, however, be passed over, as at any rate suggestive of an action of this nature.

(1.) The occurrence of deep-seated inflammations from surface chills. In most instances of this kind it is quite impossible to believe that the organs themselves are affected by the chill, as for instance when the kidneys, wrapped up as they are in their thick coatings of fat, &c., take on acute inflammatory action after exposure to a cold wind. In these cases the inflammation is probably produced through the mediation of the nervous system; and there can be little doubt that the peripheral sensory nerves take an important part in the process. As, however, it is very exceptional for the initial chill to be local in its action, it is difficult to be sure that the deeply-seated inflammation has been induced by the action of the chill on the overlying parts. We, nevertheless, tacitly by our action, admit the probability of this connection; for the man with a weak chest is warned to take especial precaution in wrapping up his chest; the man with delicate kidneys is made to wear a flannel-band round the abdomen; "cholera-belts" are served out to troops in warm climates, and so on.

There is one instance of a deep-seated inflammation occurring from a local chill which is, I believe, quite confirmatory of the views above stated. I refer to the inflammation of the facial nerve in the Aquæductus Fallopii resulting from exposure of the cheek to a draught. It is difficult to believe that the nerve lying deeply in and beneath the parotid gland can itself be sufficiently affected by the chill to induce inflammation. The inflammation is probably strictly analogous to that met with in other deeply-seated structures as the result of chill; and in this case the relation between the seat of the chill and the inflammatory effect is a very definite one. I am aware that I am entering upon debatable

ground, as to the respective parts taken by the central nervous system and sympathetic and vaso-motor systems in the production of inflammation. Nevertheless, I would offer these as suggestions, in the hope that those more competent than myself to deal with these difficult questions may be able to throw further light upon the subject.

There is one curious fact which I would mention in this connection, viz. the occurrence of duodenal ulcers in cases of surface burns. Having no good medical library within reach, I am unable to ascertain in what class of burns these ulcers have been chiefly met with. If, as I believe it has been stated, they occur in connection especially with superficial burns of the abdomen, may we not look upon their occurrence as another instance of the effect upon deeply-seated organs of superficial lesions over the organ?

(2.) I now pass to another point. If the radiation of the pain in angina pectoris, and the effects of counter-irritation over deeply-seated morbid structures, be both of them due to sensory co-ordination in the spinal cord, can we, by a study of the former, ascertain any facts which may help us in understanding the modus operandi of the latter?

It is exceedingly common to find in patients subject to attacks of angina pectoris extending to the left arm, that the arm remains in an abnormal condition for a longer or shorter time after the attack is apparently over. The most usual description is that the arm is numb—the sensation to touch is dulled. Frequently also there is a subjective "numb-feeling," i.e. not only is the sensation to touch dulled, but the patient has the positive sense of numbness—as one of my patients described it, a "buzzing" in the limb, because it seemed to him so allied to buzzing in the ears. Then again there may be the opposite condition to loss of sensation, viz. pain; either spontaneous, or developed by pressure—soreness. Thus, one of Trousseau's patients suffered from "painful numbness" in the left arm after the attacks; and on the other hand the patient who served as my text, had soreness and tenderness in the left side after his attacks.

(3.) A third variety of after-effect is the sense of weakness or "heaviness" that some patients experience in the arm. And

lastly, in one of Trousseau's patients the left arm became pallid, and subsequently of a bluish tinge.

Let us translate these various descriptions into their physiological equivalents. Loss of sensation in such a case means that the sensory nerve-centre implicated (in all probability the spinal centre) is exhausted by the nerve storm through which it has passed to such an extent, that it is no longer capable of acting fully under ordinary stimuli; and observe, that in not a few cases it is the centres to which the storm has passed from the primary source of commotion which give evidence of this great exhaustion.

The positive sensation of numbness, the "buzzing" in the limb, must mean that the commotion does not at once die down, but goes on for a time in a quiet manner; sufficient to attract the patient's attention, but not enough in most cases to amount to actual pain. The positive sensation is generally accompanied by more or less exhaustion of the centre, as is shown by the presence simultaneously of dulness of sensation to touch.

Soreness or tenderness mean that the centre is not exhausted, but is, on the contrary, left by the attack in an irritable condition, so that ordinary stimuli produce an over-action in the centre. The sense of weakness or heaviness in the limb shows that in those cases in which it is present, the whole nervous apparatus of the limb is for the time disorganised, the motor functions suffering as well as the sensory. It would be interesting to enquire into the mechanism by which this modification in the motor functions is produced, but I do not propose to do so in the present paper.

In order to apply the above rough accounts of what probably takes place, so as to give a full explanation of the action of counter-irritants, it would be necessary to have a clear knowledge of the influence of the central nervous system upon the production of inflammation. My knowledge of this branch of the pathology of inflammation is insufficient to enable me to do this, but I would make the following suggestions:—

(1.) Whatever may be the influence of the nervous system over inflammation, it is probably an *over-active* or *irritative* influence rather than the contrary.

(2.) Two methods suggest themselves to combat this over-activity. In the first place, we endeavour to soothe it; but failing in this, we endeavour to tire out the centre more rapidly than it would tire of itself.

(3.) Since in the case of deeply-seated inflammations we cannot act directly on the centre by stimuli, we act upon those centres with which it is intimately associated; stimulating these powerfully, in the expectation that they will pass on the commotion induced in them to the centre upon which we wish to act.

(4.) The centres most intimately associated with that governing the inflamed part are those controlling the tissues in the neighbourhood of the part, and hence we stimulate the surface with a view to acting on the organ lying beneath it.

(5.) The centre controlling the inflamed part being thus goaded to further action, rapidly becomes exhausted, and thus its irritative action ceases.

(6.) We have seen that even after so severe a commotion as that present in an attack of angina pectoris the centres are not always exhausted to the point of ceasing to be excitable, but on the contrary are occasionally left in a state of irritation. I believe that the analogue of this is now and then met with in counter-irritation. A patient was under my care suffering from long-standing spastic paraplegia, associated with spinal pachymeningitis. The actual cautery was freely applied on several occasions with great ultimate benefit, but the first effect of the cautery was to increase markedly the spastic symptoms (i.e. the symptoms of irritation), which after a few days diminished, until they were considerably less than before the application.

(7.) I have hitherto said nothing of the vaso-motor nerves, which play so important a part in inflammation. Without pretending to have much knowledge of the mechanism by which the vaso-motor nervous system acts in inflammation, I believe I shall be correct in saying that the influence of the central nervous system on this process is largely exerted through the mediation of the vaso-motor nerves, and the action of the latter has to some extent been presupposed in speaking

of that of the former. The profoundly intimate connection between the central nervous system and the vaso-motor system is a matter of every-day observation. In this connection the observation of Trousseau already mentioned is of great interest, viz. that in one case where the pain of angina pectoris radiated to the left arm, the arm became first pallid and then bluish. This indicates most clearly the close connection that subsists between the spinal sensory centres for a region, and the vaso-motor centres for the same region.

In the above account I am aware that I have touched upon much debatable ground; where, moreover, a far greater knowledge than I possess would be necessary to do full justice to the subject. It is quite certain that much has yet to be learnt in connection with many of the points I have mentioned, and my object in writing this paper has been to offer a few hints as to the directions in which investigation is likely to lead to successful results.

SOME STATISTICS OF CHOREA.

BY ANGEL MONEY, M.D.

Registrar to the Hospital for Sick Children.

Dr. Gowers suggested to me that in the records of University College Hospital there would be found a number of cases of Chorea Sancti Viti which had up to the present never been worked up. The readers of this article will not fail to perceive that the method so completely followed by Dr. Gowers in the tabulation of his large number of cases of epilepsy has been applied to the statistics here presented; with one or two exceptions, the shape the following tables have taken was drawn up by Dr. Gowers himself. The bulk of the cases here used come from the case-books of the various physicians, past and present, to University College; the remainder have been culled from the practice at the Hospital for Sick Children. For permission to make free with the notes, I sincerely thank the present physicians of both hospitals. I have been led by my study to form no peculiar views concerning the malady. Dr. Sturges has advanced the notion that Chorea may be thought of as a sample of *Dissolution* in the sense in which Dr. Hughlings-Jackson has used that term, and which was borrowed from Herbert Spencer.

If Dr. Sturges think that the restlessness of infancy be comparable to chorea, he must be prepared to explain away certain objections which, nevertheless, I do not say really invalidate his theory. With the roll of the tide of infant restlessness the babe gathers strength; such is not the case with sufferers from chorea. I have not the least bias against the notion that the disease is a *functional* one; but there must be an anatomical basis for chorea, just as there is a proper structure corresponding to healthy movements of all sorts. This, anatomy

has up to the present defied to observation, but it certainly exists, even if it be ultra-microscopical.

Total number of Cases.—Sex.—Age.—Number of Attacks.

The total number of cases collected was 236; of these 214 were available as regards sex, 52 were males, 162 females. The age was available in 186 cases:—1 was 4 years old, 3 were 5, 6 were 6, 15 were 7, 13 were 8, 20 were 9, 21 were 10, 20 were 11, 15 were 12, 17 were 13, 10 were 14, 13 were 15, 9 were 16, 6 were 17, 5 were 18, 3 were 19, 6 were 20, 1 was 21, 6 were 22, and there were 14 above this age. In 197 available cases there were 131 who had 1 attack, 46 who had 2, 15 who had 3, 1 who had 4, 1 who had 5, and 3 who had more relapses than this.

Month of the Year.

With regard to the month of the year when the case first came under observation, there were 30 in January, 16 in February, 23 in March, 27 in April, 20 in May, 25 in June, 11 in July, 11 in August, 11 in September, 17 in October, 16 in November, 17 in December; these, re-arranged according to quarters, give 69 for the first quarter, 68 for the second, 33 for the third, 50 for the fourth. It is interesting to remark the great falling off during the warmer months of the year; what the explanation of this is, however, is not to be too hastily concluded.

Rheumatism.

Of 214 available, there was a history of genuine rheumatic fever in 33, of *rheumatism* in 23, and of very doubtful rheumatic history, 9; so there was an undeniable rheumatic history in about 16 per cent., a possible rheumatic history in about 29 per cent., and the utmost that can be made of rheumatism is 32 per cent.

Heart-disease.—So-called functional murmurs.

There was real heart-disease in 31 cases, or about 13 per cent.; 29 were cases of mitral disease, 2 of aortic regurgitation; no distinction is drawn betwixt mitral obstructive and mitral regurgitant, purposely. There was a systolic murmur

in 64 cases, or about 23 per cent.; this was apical in all the cases except 11.

Cause assigned.

The cause assigned (214 cases available) was fright in 60 cases; accident in 13 (a distinction of doubtful value has been made betwixt psychical and physical shock); hard school-work in 9 (none of these cases come from the University case-books); anxiety and worry, 6; imitation, only 2; want of food, 2; sore thumb, 1; exposure, 1; no assignable cause, 120.

Heredity.

With regard to Heredity (214 available), there was a rheumatic history in 28: 9 times the father was rheumatic; 9 times the mother; 3 times the father and mother; once the mother, brother, and sister; once mother and brother; once father, brother, and sister; once mother and mother's brother; once the grandmother; twice the brother. There was a history of chorea in 14: 3 times the father; thrice the brother; thrice the sister; once the sister and cousin; once the niece; once the mother, father, and brother; once the mother's sister; once the mother's brother. There was a history of fits in 6: twice in the mother, once in the father, once in the brother, once in the sister, once in the father's mother. There was a history of rheumatic gout in 5; 4 times it was the father, once the father and father's mother. There was a history of megrim twice; once in the mother, once in the father and others. Gout was recorded twice; once with the father, once with the aunt. Epilepsy was noted twice; once in the father, once in the uncle. Convulsions were registered once in the brother; once the brother was an imbecile, once the brothers and sisters had suffered from "head affection," once the father had delirium tremens, and once the mother was a drunkard. Several of these collateral diseases were thus associated: once the father had rheumatic gout and rheumatic fever; once the father had rheumatic gout, and the mother and brother had rheumatic fever; once the father had rheumatic gout and epilepsy; twice the father had had rheumatic fever and chorea; once the father and mother had had rheumatic fever and the brother chorea; once the father had rheumatic fever and the niece chorea; once

the mother had rheumatic fever, and mother, father, and brother had chorea (this and the two next are highly pertinent); once the mother had rheumatic fever and her sister chorea; once the father had had chorea and the brother was insane.

Starting-place.

The chorea was said to have begun 6 times in the right hand, 6 times in the right arm (=12 times in right upper limb); 6 times in the left hand, 6 times in the left arm (= 12 times in left upper limb); once in the right leg, twice in the left leg, twice in the left face, 3 times in the right arm and leg, once in the left arm and leg, thrice in the head, twice in the speech, once in the legs, once in the arms, once in the speech and hands. These are the only cases out of 214 in which the site of origin of the disturbance was given.

The chorea started 33 times in the right side, 30 times in the left; it was worse 55 times in the right, 49 times in the left. There was hemichorea 3 times in the right, and 4 times in the left.

The march of the Chorea.

In 24 cases a description of the progress of the chorea from part to part was given. In one case the movements began in the right arm, then went to leg, then to left leg and arm; in another it commenced in the left arm, then turned to leg, then to right hand, and on to right leg and face; in another, it first struck the left leg, then affected speech, and lastly, both hands; in another, the left face was first affected, then the hand and leg, and afterwards the right side and speech; in one case the head first moved, then the left hand, left leg and right arm in succession; again, it was the left arm and leg before the right; in another, the right arm and leg first suffered, then the speech; in one, the right-hand disturbance was followed by that of the arm, leg on the same side, and then the opposite arm and leg; in the ninth instance, the left hand was the first troubled, then the leg, and then the face; in the next, it was the left arm, and afterwards the leg; in the next, the right hand shook first, then the right leg, then the opposite limbs; in another, the left arm was jerked, and later, the face, and latest, the leg; yet again, it was the legs which

first shuffled, then the hands, and then the arms; in another instance, the right arm was first fidgety, then the right leg; in the fifteenth case, the left hand first fumbled, then the arm, and then the legs were affected; in the next, the right hand first dodged about, then the leg, and then the left side; in another, it was the head and then the arms; in another, the left face twitched, and afterwards the left arm and leg; again, it was the right brachial which preceded the right crural disturbance; in yet another, the arms were first awkward, then the legs, and finally the head; the right leg, arm, face, and left side, was the order of succession in another; the head, hands, feet, in another; the left limbs before the right in the last but one; and in the last, the passage was from left leg to left arm, to left face. These are the only cases which describe the course of the chorea from part to part. I may as well state, once for all, that the material here used is not to be implicitly relied on; but, I think, it may be used with a certain amount of trust and reserve. One thing seems to come out of this small number of cases, viz. that when the disturbance of motion starts unilaterally in a particular region, it will travel over the whole of that one side ere it pass to the opposite. Generally speaking, my notes show that the side of the body first affected was the worse; but this was not always the case.

Duration.

In 19 cases the affection lasted 1 month; in 66, two months; in 33, three months; in 18, four months; in 7, five months; in 9, six months; in 1, eight months; in 1, nine months; in 1, eleven months; in 5, one year; in 4, two years; in 1, three years; in 5, *many years*. The above estimate is probably in many instances too short, as many patients left the hospital still showing some movements.

Duration qua *Age.*

The patient being under 7 years of age, 4 cases lasted two months; and 4, three months: the patient being 7 years old, 1 case lasted one month; 7 cases, two months; 4 cases, three months; 2 cases, four months. These statements will serve to explain the following table:—

Duration.	Age in Years.												
	Under 7.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	−20.	20 +
1 Month ..	..	1	1	1	3	1	2	2	..	2	1	4	..
2 Months ..	4	7	3	6	4	6	5	5	7	5	4	10	2
3 „ ..	4	4	5	7	5	4	3	2	2	1	2	3	2
4 „ ..	..	2	1	3	3	2	2	1	2	3	1	..	..
5 „ ..	..	..	..	1	..	..	2	2	..	2	..	..	..
6 „ ..	..	..	1	..	..	3	..	1	..	1	..	..	..
Over 6 Months	..	..	..	..	..	..	1	1	..	..	..	..	..
Over 1 year ..	1	..	1	1	3	3	2	1	1	..	..	1	..

Duration qua *Fright.—Rheumatism and Heart-disease.*

In 8 there was a history of fright, rheumatism, and the patient had heart-disease, and the disease lasted under three months; in 1, with a similar history, the disease lasted under six months; in 1, with the same history, it lasted over six months, but under a year.

In 6 there was a history of rheumatism, and the patient had heart-disease, but no fright, and the malady was over in less than three months; in 5, with same history, it endured under six months; in 1, under same history, it continued over six months.

In 10 cases there was a history of rheumatism and fright, but no heart-disease, and the affection lasted under three months; in 4 it continued for less than six months.

In 23 cases with rheumatic history alone, the disturbance lasted under three months; in 5, less than six months; and in 1, longer than six months.

In 7 instances where heart-disease only existed, the chorea was over in less than three months, and once only lasted less than six months. In 2 cases where there was a history of fright with heart disease, the chorea finished up in less than three months. In 21 cases in which only fright existed in the history, the disease lasted less than three months; in 8, it went on for less than six months; and in 4, for over six months.

In 51 cases in which there were neither heart-disease, nor rheumatism, nor fright, the malady ran its course in less than three months; in 15, in less than six months; and in 1 in less than a year.

Intervals between Relapses.

Five times the interval between the first and second relapse was under half a year; 5 times it was half a year; 13 times, one year; 15 times, two years; 3 times, three years; 5 times, four years; twice, five years; once, six years; twice, eight years = 51 cases in which relapse occurred once. Three times there was an interval between the second and third attacks of less than half a year; 3 times half a year, 6 times of one year, 4 times of two years, once of five years, once of six years = 18 cases of a second relapse. Once there was an interval of half a year between the third and fourth attack; once of a year, twice of two years = 4 cases of a third relapse. Once there was an interval of less than half a year between the fourth and fifth attack; once of half a year, once of a year = 3 cases of a fourth relapse.

Intervals after Fright.

The St. Vitus's dance began immediately after the fright in 9 females and 1 male = 10. Two of these began in the right side, and 1 in the left (the rest not available in this regard). The chorea began one day after the fright in 3 female cases; it began two days after in 1 female case; it began one week after in 1 female and 1 male case; two weeks after in 2 females, three weeks after in 1 female, one month after in 2 females.

TABLE SHOWING THE RELATION OF MALE AND FEMALE CASES WITH ANY COMBINATION OF RHEUMATISM, FRIGHT, AND HEART-DISEASE. R = Rheumatism; F = Fright; H = Heart-disease; O means nothing; M = Male; F = Female.

AGE.	R. H. F.		R. H. O.		R. O. F.		R. O. O.		O. H. O.		O. H. F.		O. O. F.		O. O. O.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Under 7	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	5
„ 7	..	1	..	..	..	..	..	1	..	2	..	..	..	1	..	7
„ 8	..	1	..	1	..	..	..	1	..	..	..	..	..	2	1	6
„ 9	1	..	..	1	..	..	..	1	..	1	..	..	..	1	3	5
„ 10	..	..	..	1	..	2	2	2	..	1	..	..	..	3	1	3
„ 11	..	1	1	..	1	2	1	2	1	..	..	..	..	3	1	4
„ 12	1	..	..	1	..	2	1	2	..	1	..	..	..	1	2	7[1]
„ 13	1	..	..	1	..	..	1	2	..	1	..	1	1	4	1	3
„ 14	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	5
„ 15	..	..	..	..	..	..	1	3	..	..	..	..	..	3	..	7
„ 16	1	..	..	..	..	1	1	2	..	..	..	..	..	3	..	..
„ −20	..	..	..	..	..	3	1	3	..	1	..	1	..	2	..	7
„ 20+	..	..	..	..	..	..	..	2	..	..	..	..	..	6	4	6

[1] As an example, it will be understood that 7 females of the age of 12 had no heart-disease, no history of fright or rheumatism.

TABLE showing the Month at which the Disease came under Observation, and the Presence or Absence of Heart-disease and Rheumatism. The letters mean as in the previous Table.

R. H.		R. O.		H. O.		O. O.		
M.	F.	M.	F.	M.	F.	M.	F.	
[1]2	1	1	4	..	2	1	17	January.
1	..	..	1	..	..	3	7	February.
..	2	..	7	..	..	1	13	March.
..	2	4	4	..	1	5	10	April.
..	2	..	3	..	..	2	7	May.
1	..	2	2	..	..	1	10	June.
..	1	..	..	..	3	1	6	July.
..	..	..	1	..	..	4	5	August.
..	..	3	1	..	..	2	3	September.
2	2	..	3	..	..	1	7	October.
..	..	1	2	..	..	4	7	November.
1	1	..	2	..	..	3	10	December.

Re-arranged thus—

[2]6	13	2	42	1st quarter.
5	15	1	35	2nd „
1	5	3	21	3rd „
6	8	..	32	4th „

[1] *E.g.* 3 cases (2 males, 1 female) who had heart-disease and had had rheumatism were affected in January.

[2] There were 6 cases with heart-disease and rheumatic history affected with chorea in the 1st quarter of the year.

TABLE showing Number of Cases with or without History of Rheumatism and Fright, qua Age and Sex.

	R. F.		R. O.		F. O.		O. O.	
	M.	F.	M.	F.	M.	F.	M.	F.
Under 7 Years ..	..	..	..	..	..	2	1	5
„ 7 „ ..	..	..	..	1	..	1	..	9
„ 8 „ ..	..	1	..	1	..	2	1	6
„ 9 „ ..	..	1	1	1	3	1	4	6
„ 10 „ ..	..	2	2	3	3	3	1	3
„ 11 „ ..	2[1]	2	1	3	1	3	1	4
„ 12 „ ..	1	2	2	2	1	1	2	7
„ 13 „ ..	..	1	2	2	..	5	1	4
„ 14 „ ..	..	..	1	..	1	1	1	6
„ 15 „ ..	..	..	1	3	..	3	..	8
„ 16 „ ..	..	1	2	2	..	3	..	1
„ −20 „ ..	..	6	1	3	1	2	..	4
„ 20+ „ ..	..	..	..	1	..	5	..	2

[1] *E. g.* there were 4 cases (2 males, 2 females), aged 11 years, who had a history of both rheumatism and fright.

Heart-disease: Time; Relation to Chorea.

The heart-disease preceded the chorea certainly in 7 cases (6 mitral regurgitation, 1 aortic and mitral regurgitation); in 18 cases it was doubtful; it was probable in 5 cases (mitral regurgitation, 4; double aortic, 1); the heart-disease appeared after the chorea in one case.

Qua *side.*

In 8 cases the right side was affected when the patient had heart-disease; in 7 cases the left.

When the heart-disease certainly, or probably, preceded the chorea, the right side was affected thrice, the left twice.

(Only 5 out of the 12 cases show which side was most affected.)

At different Ages.

From five to ten years of age there was heart-disease in 12 cases, no heart-disease in 56.

From eleven to fifteen years of age there was heart-disease in 12 cases, none in 67 cases.

Over fifteen years of age the heart was diseased in 7 instances, not diseased in 45. The heart was affected therefore 31 times out of 168 available cases.

Qua *Relapse.*

There was a relapse of the chorea without the presence of heart-disease in 48 cases; there was no relapse with the presence of the heart-disease in 17 cases; there was relapse and heart-disease, 14 times.

Fallacies here are great—e.g. one cannot say whether a relapse might not occur.

Fright qua *Relapse.*

There was a relapse of the chorea and a history of fright in 25 cases; there was a history of fright and no relapse in 42 cases; there was a relapse and no history of fright in 41 cases.

Rheumatism qua *Relapse.*

There was a relapse and a history of rheumatism in 19 cases; there was a history of rheumatism and no relapse in 45 cases; there was a relapse and no history of rheumatism in 45 cases.

In thus presenting these statistics to the medical profession, I am well aware that they may be thought to be very uninteresting, that is because I have not been able to draw from them any very notable general principles; but they are of value, if only in a negative way, and this must be my reason for publishing them.

HEMIPLEGIC MUSCULAR ATROPHY OF PERIPHERAL ORIGIN.

BY DAVID FERRIER, M.D., F.R.S.

Physician to King's College Hospital, and the National Hospital for the Paralysed and Epileptic.

APART from mere functional disturbances, either of exaltation or depression, to which many convulsive and paralytic symptoms may be ascribed, resulting from peripheral lesions of nerves, it is established that lesions of nerves may also induce anatomical changes in the nerve-centres. And the experimental induction of such changes promises to be one of the most reliable methods of determining the position of the various sensory and motor centres and tracts in the brain and spinal cord. It has been shown by the investigations of Dickenson, Vulpian, and others, that as the result of section of nerves, and in cases of long-standing amputation or congenital absence of a limb, certain centres and tracts in the spinal cord and brain undergo atrophic degeneration. In these cases there seems to be mere simple atrophy, without indications of inflammatory action. In the spinal cord the wasting is visible both in the white matter, particularly the posterior columns, and also in the grey substance corresponding to the attachment of the nerves in question. In the cerebral cortex, also, atrophy has been observed in the motor regions, corresponding to those movements which have been long lost.

The effects on the nerve-centres of irritation or inflammation of peripheral nerves are, however, of much greater pathological importance. Under this head come the so-called reflex paralyses, the pathology of which has been the subject of much discussion. It has been found experimentally, and also in man, that in connection with severe injuries of viscera and

traumatic lesions of nerves, paralysis may occur in regions not directly related to the nerves actually injured. These paralyses come on at once, and subside mostly with the irritation which has induced them. Many such facts have been recorded by Weir-Mitchell, and others. It is obvious in such cases that the paralyses are of a purely functional nature, as they occur before any organic changes could possibly be induced in the nerve-centres. What is the exact nature of the functional disturbance is a matter more of speculation than actual demonstration, and the theory of vascular spasm of Brown-Séquard, or the theory of exhaustion of Jaccoud, may be regarded as more or less satisfactory.

It is otherwise, however, when the symptoms of morbid affection of the nerve-centres do not show themselves at once, but at some considerable interval after the infliction of the injury. The cases of reflex paraplegia which have been so often seen in connection with affections of the bladder and prostate in man, and the uterus in women, seem to be of a different type from the above; and the views that the paralytic affections in these cases are not merely functional, but due to actual organic changes in the spinal cord, propagated along the nerves of the diseased viscera, have received much support from experimental and pathological investigation of recent years.

Inflammation of peripheral nerves induced in the lower animals by irritants, mechanical lesions and the like, has by some observers been found to be altogether negative as regards propagation to the spinal cord. But others (Hayem, Klemm, Niedieck, &c.) have described both meningeal and myelitic inflammation as the result of such lesions. The resulting neuritis, which, according to Hayem, is both interstitial and parenchymatous, ascends or descends from the primary lesion not in a continuous manner, but interruptedly—*neuritis disseminata vel migrans*—portions of apparently healthy nerve alternating with inflamed portions. In this manner the neuritis reaches the cord, and induces either meningeal inflammation from the direct continuity of the spinal membranes with the neural envelopes and interstitial tissue, or a poliomyelitis with atrophy of the multipolar cells, of greater or less

extent and acuteness, probably through the medium of the posterior roots.

These experimental data render it probable, if it has not been actually demonstrated, that similar processes underlie certain symptoms which are occasionally seen in man in connection with peripheral nerve lesions. It has been found that at more or less distant intervals of time after traumatic lesions of nerves, articular inflammations, and amputation of limbs, atrophic paralysis or muscular atrophy has occurred in regions not directly related to the nerves primarily injured. Thus, after an injury to the ulnar nerve, as in a case reported by Brown-Séquard, muscular atrophy showed itself in the region of distribution of other nerves of the brachial plexus, in a manner which could not be accounted for by mere extension by contiguity. Many other instances are on record of atrophy of a limb from injury of one of the branches of the plexus supplying it.

Not unfrequently, also, atrophic degeneration occurs in the opposite limb, presumably from a transverse extension of the myelitic process; and there are instances, also, which would seem to show that the myelitis may be propagated longitudinally in an ascending or descending manner, so that atrophy occurs in both the limbs on the side of the original lesion.

Cases of this kind have been recorded by Vulpian, Charcot, Leyden, Terrier, and others.[1]

In addition to those already recorded, I may mention the following case, which has just come under my observation.

A young man, aged 27, who had left hip-joint disease in childhood, and had recovered with a stiff and atrophied limb, with which, however, he was able to support himself and walk, a few months ago rapidly lost power in this leg, so that now he is unable to support himself with it. A few weeks afterwards he began to experience numbness and weakness in his left hand, and lately he has experienced numbness in the ulnar region of the right hand also.

On examination, the interossei of the left hand were found

[1] See an able summary and review of facts bearing on this question by Ch. Talamon, 'Revue et Mensuelle de Méd. et Chir.,' July 1879, *et seqq.*

to be considerably wasted, the basal phalanges hyper-extended, and the use of the fingers characteristically impaired. The wasted muscles showed well-marked reaction of degeneration.

Though the right lower limb was apparently unaffected, yet there was greatly increased knee-jerk, and tendency to ankle-clonus. The power of retention of urine had also become considerably impaired since the symptoms first developed themselves.

In this case there seems to be both an ascending and transverse myelitis of a subacute form, the starting-point of which is probably the articular inflammation of the left hip, and the atrophic changes in the lumbar region of the spinal cord, which have long remained quiescent. The patient dated his symptoms from the use of a new boot which he had himself constructed, and the use of which in walking seemed to cause unusual fatigue.

A truly hemiplegic form of muscular atrophy has very rarely been observed. Leyden, in his work on 'Diseases of the Spinal Cord,' reports a case of a gunshot injury of the left thigh which was followed by severe pains in the leg, and afterwards in the left arm. Nearly three years after the receipt of the wound the patient became paralysed or paretic on the left side, leg, arm, and face. The acuteness of vision also of the left eye was impaired. Examined by Leyden in Jan. 1874 (the wound having been inflicted in Nov. 1870), the patient was weak on the left side, and complained of a feeling of heaviness in it. The muscles generally on the left side were flaccid and less voluminous than those of the right, and the electric contractility was somewhat diminished, and abnormal.

The following case which I have had under my care shows by its history and progress the recurrence of a truly hemiplegic muscular wasting, with paræsthesia, in connection with neuritis of the stump of an amputation of the left hand at the wrist.

The patient, ætat. 28, is a member of the medical profession; and he thus described his case. While shooting in India one day in June 1877, his gun burst, inflicting a scalp wound, also rupturing the left membrana tympani and

seriously lacerating his left hand. The hand was at once amputated just above the wrist, and in a little more than three weeks he was able to return to duty. The stump had almost entirely healed, and the scalp wound and laceration of the tympanum had quite healed up.

For two years subsequently to the amputation he experienced almost daily pain in the stump. The pain varied in character. He described it as like that caused by tension of the flaps over the face of the stump; as a burning sensation; as if something were contracting and subjecting the ends of the nerves to pressure; as a throbbing sensation.

Later on, this condition of the arm appeared to be greatly influenced by changes in the weather. A hot and dry climate was most agreeable, whereas a sultry and moist atmosphere was always the cause of pain; as also cold and frosty weather.

Soon after the operation he observed wasting of the left arm.

Towards the end of 1878 he began to feel as if a piece of parchment were drawn tightly over the left side of his face, which was so unpleasant that he frequently tried to relieve it by rubbing.

He also felt "unbalanced" or "lop-sided," which was particularly disagreeable. This feeling of "lop-sidedness" grew worse about August 1879, and at this time he observed some wasting of his left leg. Later, he noticed a similar wasting of the left side of his thorax and abdomen.

All these symptoms the patient noticed before they could be recognised by others.

During the attacks of pain in the stump the face always felt more drawn than at other times. One night after his return to England, at the end of 1881, during a severe attack of pain, he felt as if there were a muscular thrill through the left side of his chest. For the last year there have been sensations of pins and needles in the left foot, and fibrillar twitchings in the left leg and thigh. Similar twitchings have also been felt in the left side of the face, thorax and abdomen. He now—January 1882—feels as if there had been a layer cut off the whole of the left side of the body, and complains much of his "unbalanced," "lop-sided" sensation.

The patient was a well-developed, vigorous and healthy-looking man. All the organic viscera and functions were normal. It was plainly evident on examination of the two sides of the body that there was a want of symmetry, but the respective measurements were not determined at this time. The muscles of the left side reacted well to the faradic current, and no very evident difference could be discovered between those of the right and left side.

Sensibility was normal.

The question of re-amputation was discussed, and the patient further consulted Mr. Lister, who advised this at once. The stump was re-amputated by him on January 28, 1882, and the healing of the wound proceeded satisfactorily. A few days after the operation, pain recurred in the stump, accompanied by twitchings of the arm, and tenderness, with hyperæsthesia to cold over the whole of the left side.

After that, the patient ceased to complain of attacks of pain, and the paræsthesia of the left side of the body somewhat diminished. But the feeling of "lop-sidedness" still continued.

On February 22 I made comparative measurements of the two sides of the body.

By stretching a tape-measure between fixed points on the two sides of the face in various diameters, there appeared a difference of one-eighth of an inch in favour of the right side—

		in.			in.
Widest part of thigh . .	Right	= 21⅜	..	Left	= 20⅜
Four inches above patella .	Right	= 15½	..	Left	= 14⅜
Calf of leg	Right	= 13⅛	..	Left	= 13 1/16
Abdomen—two inches above umbilicus	Right side	= 16⅜	..	Left	= 16¼

The arms were not strictly comparable for obvious reasons, but the circumference round the acromion and axilla was one inch less on the left side than the right.

There was thus an appreciable difference between the right and left side of the face, trunk and limbs, varying from one-eighth of an inch on the face, to one inch in the limbs.

The patient shortly after this put himself at my advice under the care of Dr. de Watteville for electrical treatment.

This was carried out by him for about six weeks, by his method of galvano-faradisation, which he has recently described in the 'Neurologisches Centralblatt' for June 15, 1882.

Under this treatment the patient gradually improved in all respects, and on my again taking the same measurements on May 23, I found that there was now no appreciable difference between the two sides of the face. The widest part of the right thigh now exceeded that of the left by only one-eighth of an inch; the right calf measured only one-eighth more than the left; and similarly as regards the two sides of the trunk.

The patient ultimately returned to his duties in India, to all appearance perfectly recovered.

The above particulars show an evident causal relationship between the neuralgic stump of the left hand, and the occurrence of the peculiar "lop-sided" paræsthesia of the left side, and actually demonstrable wasting of the face, trunk and limbs on that side, which speedily ceased after re-amputation of the stump, and properly directed electrical treatment.

The condition of the nerves of the stump was examined by Mr. Watson Cheyne, who has furnished me with the following account of his examination.

"Examination of the stump immediately after amputation showed the presence of a firm neuroma, about the size of a small hazel-nut, affecting the divided end of the radial nerve. The nerve itself was thickened, and had a distinctly rosy hue, one or two vessels being also evident in its sheath. The lower end of the ulnar nerve was somewhat expanded, but this expansion did not present the distinct form of the radial neuroma, nor was it so firm. The nerve was of the normal white appearance. The median nerve was apparently quite healthy.

"*Microscopic characters.*—Longitudinal and transverse sections were made both of the neuromata and of the trunks of the radial and ulnar nerves. The neuroma at the end of the radial nerve was found to consist of a large amount of fibrous tissue, enclosing in small alveoli bundles of degenerating

nerve-fibres, in some places indeed the arrangement of the fibrous tissue showed that nerve-bundles had already disappeared. Here and there the fibrous tissue was still almost embryonic. In the trunk of the nerve there was great increase of the fibrous tissue between the bundles of the nerve-fibres which were undergoing degeneration. There was a considerable number of small blood-vessels, some of them surrounded by embryonic cells. This appearance was especially marked towards one side of the nerve, where there was a distinct line of granulation tissue. In the ulnar nerve there was thickening of the fibrous septa to some extent, but not nearly so marked as in the radial; there was no appearance of granulation tissue either in the trunk of the nerve or in the enlargement at its end.

"The appearances indicate chronic inflammation of the radial nerve, both at the divided end and along the trunk. This process was still going on at the time of the operation, as evidenced by the granulation tissue found along one side of the nerve trunk. There was no evidence of continuance of inflammation in the ulnar nerve."

Whether in this case there was any morbid change in the anterior grey column of the spinal cord of the left side capable of histological demonstration, or whether it was merely a so-called functional depression due to the long-standing peripheral irritation, can only be a matter of speculation. The latter view might be considered as the more probable from the fact of recovery on cessation of the irritation, though this would not necessarily follow.

The point of importance is the possibility of a morbid condition of the whole of the grey column of one side of the spinal cord being developed in connection with long-standing peripheral irritation, ascending and descending from the point of primary attachment of the inflamed nerve.

Critical Digests and Notices of Books.

Leçons Cliniques sur des Maladies Mentales et sur les Maladies Nerveuses, professées à la Salpêtrière. Par le Docteur AUGUSTE VOISIN, Médecin de la Salpêtrière. Paris, Baillière et fils. 1883. 8vo.

LECTURES are perhaps not strictly clinical when they are delivered in the theatre of an hospital, and are not illustrated even by reference to cases actually under treatment. Yet these lectures of M. Voisin, replete as they are with the records of illustrative cases, strike one almost with the vividness of bedside teaching. Moreover, we learn from some casual remarks, that on one day of each week a limited number of the students are admitted within the wards of the great asylum, to observe for themselves the phenomena of mental disease. The authorities of the Salpêtrière take a middle course between the free admission of medical observers, which some people think injurious to patients within an institution, and the practical exclusion of unofficial medical men, which more certainly, by perpetuating professional ignorance of insanity, is, and must be, detrimental to that larger body of the insane who have not been placed under such treatment as that of the hospital of the Salpêtrière.

The difficulty of providing real clinical instruction in mental disease, that is to say, instruction illustrated by actual cases, for the vast number of recruits which the great profession of medicine demands, is perhaps so great that it may never be overcome, and so the public will in time become aware that the bulk of medical men may know as little from observation about lunatics as living priests know about the angels whom they have never seen. In this country the promising commencement of such teaching has all but withered in the bud. In France, however, as we see in this publication, it is still

attempted by means of lectures, more or less illustrated by examples, and such lectures, if they fail to attain the highest value of teaching by demonstration, are of great worth, especially to those fortunate practitioners of the medical art who are not compelled to draw entirely upon the resources of their imagination for some kind of picture of the lunatics they hear about. The resources of photography are but a feeble aid, except to the initiated. M. Voisin gives several pages of excellent photographs; but to any one who has never learned to read the lunatic feature and figure, we venture to think that they will convey little meaning, except of strong variations of temper and of good or bad looks. To the thousands of medical students who have never seen the play of a lunatic's features, or listened to the accents of his voice, even such instruction as that contained in our author's pages must, to a great extent, remain incapable of being appreciated; and until some plan of oral teaching, duly combined with actual demonstration, be devised and diligently carried into effect, it is likely that the early stages of insanity will remain practically unobserved or lamentably misunderstood, that the safety of the public from the insane who are at large will continue to be unguarded, and that the insane who are not permitted to remain at large will continue to be deprived of their liberty on judgments which are erroneously supposed to be the results of medical science.

We raise this lament, not over M. Voisin's important work, imperfect as his clinical demonstration appears to be, but because his work so far excels anything which we are likely to have in this country, where the neglect into which the teaching of this great department of medicine has fallen is so great that no one could be found in a position corresponding to that of this eminent Frenchman, with his materials for teaching, and especially with his audiences to be taught. M. Voisin has done well; no doubt he might do better if he could bring his students to more intimate observation of his patients. In this country the teaching of mental disease is, with one or two exceptions, either neglected or pretended. The medical men to whom the custody of the insane in ever-increasing agglomeration of numbers is entrusted, are losing the character of

physicians in that of administrators, and medical treatment for the cure of mental disease in the very institutions supposed to be devoted to the purpose seems to be passing out of use.

The first Lecture on Classification will serve as well as any subsequent one to elicit the criticism that these lectures are wanting in method, that their matter is less regular in arrangement, or, to use a homely phrase, less cut-and-dried than is usual in medical writings. We are inclined to accept this quality of the author's instructions as not altogether a disadvantage. The mass of observations on such a subject as insanity are so heterogeneous and vast, that it is impossible to reduce them to strict order, except at the expense of truth. There will be a time for method later on, when the true generalisations have been seized, upon which alone the facts can be rightly marshalled. But there is also in the development of any complicated science as it struggles out of ignorance, a time when facts and thoughts about them are grouped, and best grouped, in much detachment; and thus it is that we are far from finding fault with the somewhat rambling manner in which M. Voisin treats the various subjects in which he instructs us, leaving us not uncertain as to the main direction of his line of thought, but at liberty to feel ourselves by no means bound either to agree or to disagree with him absolutely. In a difficult country we follow a leader with all the more confidence, when we perceive that he is willing to wander from a straight line by practicable deviations.

With regard to classification, we quite agree with the author in the belief that the classification of *délires* is insufficient.

We also entirely concur in the remarks made upon the new classification attempted by the late M. Morel, and which has been imitated and exaggerated by certain writers on lunacy on this side. He says:—

"Morel, of Rouen, had much felt the defect of the actual classification, since he attempted to found one upon etiology; but for want of a sufficient number of pathogenic and anatomo-pathological facts and observations his new classification was deficient in solid foundation; it sinned also on an important side, by tending to confound forms altogether different, when it admitted hereditary insanity, for there are few mental aberrations in which one does

not find morbid heredity. By classifying in the same category all cases of alcoholic insanity one conjoins cerebral and meningeal hæmorrhages, fatty degeneration and chronic meningitis. The same difficulty presents itself in the hypochondriasis which one meets with in simple insanity and in general paralysis. I think, therefore, that it is necessary to reject all classification founded upon etiology alone. As in all branches of medicine, so progress in mental pathology is only possible by the participation of anatomical pathology, and every classification aiming at being rational, must be established upon etiology, pathogeny, clinical and anatomical pathology."—Pp. 15 and 16.

How different are these ideas from those which have been so earnestly pressed upon the profession by the Scotch school of lunacy, which is in so many other respects so practical and progressive, but which in this respect has developed the indefensible project of M. Morel into a fantastic system of etiological classification, which must, indeed, have brought down the animadversions of our author had he known of it! It would seem that M. Voisin has escaped the task of demolition in this instance by his ignorance of English. Apparently he is not acquainted with German either. Probably there is enough nonsense written in his own language for him to refute, but the literary knowledge which he possesses of his subject is very far from being cosmopolitan, and perhaps it would not be unjust to call it narrow.

As a partial classification, he divides *délire* into partial, general, and complex. We have not attempted to translate the word *délire*, which he uses, we believe, as almost tantamount to insanity; a sense commonly given to it by his countrymen, but which certainly does not correspond with our own use in medical literature of the word *delirium*. An English physician hearing that a person was delirious would be apt to conclude that he was rambling in thought in an unconscious or semi-conscious state of febrile or cerebral disease, and he certainly would not conclude that he was insane; but our professional brethren in France apparently apply the term to all insane deviations from reason in thought, and especially to delusion. The curious derivation of the word *delirium* from *de lira*, out of the furrow, seems to justify its French use as expressive of any morbid deviation from the straight and

right line of sound-minded reason. Might we not with advantage adopt this word from the French into our medico-psychological vocabulary, replacing by it the word "delusion," excepting where the latter is really appropriate, which it is not twice out of three times where it has come to be employed?

M. Voisin has not been proof against the temptation of formulating a classification of his own. He says that—

"Every rational classification must be founded on the *ensemble* of notions given to us by the symptoms, the pathogenesis, the pathological anatomy, and the etiology.

It is thus that I shall divide insanity into six classes.

1st. *Folie acquise*, that is to say, which comes on in the course of life and has been preceded by a state of reason and intelligence.

2nd. *Folie native*, a form in which the intellectual troubles manifest themselves at the earliest age, especially under the influence of heredity.

3rd. *Folie par intoxication et par virus*, the nature of which is clearly indicated by the name, or by alteration of the blood, or by diathesis.

4th. *Le Crétinisme, l'Idiotie, l'Imbécillité.*

5th. *La Paralysie générale.*

6th. *La Démence sénile.*"

The scheme is far from being unobjectionable, for is not the third class included in the first, and the fourth in the second, and why should senile dementia, so variable in its antecedents, form a class apart? Are the cases of the first, second, and third classes distinguishable either by their symptoms or by their pathological anatomy? No doubt the distinction of having been acquired from what the author calls native insanity is important, being, it would seem, the distinction which we have ourselves insisted upon between what we have called developmental insanity and accidental insanity, but it seems to us scarcely a distinction which it is wise to select as the principal basis of a system of classification.

The interesting lecture, being the thirteenth of the series, on Insanity caused by the siege of Paris and the Commune, throws light upon what the author means by acquired, as distinguished from native insanity. It had been remarked, and, if we remember rightly, an article had been written to

show, that there was no increase but even a diminution of recent cases of insanity in Paris during the fearful ordeals to which the population had been subjected from July 1870, to June 1871. M. Voisin, however, thinks that the allegation having been made upon statistics drawn from establishments which only receive patients from the rich and easy classes of society is untrustworthy, and that the poorer and labouring classes did suffer mentally, and in great numbers. However, he does not attempt to prove his case by means of statistics drawn from establishments which receive only the insane of the suffering classes, and the cases of individual suffering which he adduces, however interesting, are liable to be misleading, for it is all but impossible in any individual case to prove that there was no predisposition. It is not by heredity alone that the predisposition to insanity is formed, and when one person out of many succumbs to influences which act with equal force upon all, the inference must be that there was a predisposition to be so affected. It would seem that most men are so constituted that all the horrors of the 'Pilgrim's Progress,' or of Dante's 'Inferno,' could not drive them mad. Others there are who become insane upon severe exciting causes acting upon the mind, or upon slight ones, or upon none at all which can be discovered. While, on the other hand, it may safely be affirmed that there can be no man born of a woman who is not liable to become a madman from accidental causes of a physical nature, traumatic or toxic. Blows on the head and starvation must have caused a certain number of insanities in persons not predisposed; but it is by no means certain that the psychical horrors of war must have had any such effect. Moreover, it would seem more likely that men of leisure, culture, and opulence would be more affected by national disgrace and defeat, by social anarchy and the destruction of property, than the labouring, struggling, and suffering classes of society. M. Voisin therefore delivered an eloquent and interesting lecture, but he did not prove his case, nor upset the statistics of Charenton. The lecture also seems to indicate that the author is by no means clear as to what he really means by the somewhat taking terms of his classification, *Folie acquise* and *Folie native*. That which we have to

repeat in the matter is that there is a type of man, often enough of a sane family, who will become insane, and nothing can prevent him. His disease we call developmental insanity. Also there is another type of man whom nothing can make insane short of a physical injury affecting the brain in a certain manner, as by a syphilitic neuroma, or by poisoned blood, or some such physical interference with the organ of mind. Insanity in such a man we call accidental insanity. The two types must be recognised, but between them there is an infinite variety of susceptibility and of insusceptibility, and therefore we venture to think that this leading notion of our author's lectures ought only to be accepted with the reservation that it shall not be allowed to explain more than it is able to make really clear. The idea upon which M. Voisin founds his classification is a true one, but it is too diffuse to be of such use. Like flood-water, it will not turn a mill.

Our space limits will not permit even an appreciative reference to all the contents of the author's substantial volume of well-nigh eight hundred pages. Suffice it to say that, while some lectures are devoted to forms of insanity whose real existence we are fain to dispute, a larger and more valuable part of the volume discusses very fully, and with an ample supply of illustrative cases, such important topics as epilepsy, idiocy, general paralysis. The lectures on this last subject occupy 164 pages, and, in themselves a treatise, are too valuable and important to be dealt with in a brief critique. We may remark, in passing, that the author is inclined to call forms of disease, especially consecutive forms, by the name of general paralysis, where identity with the disease to which we in this country restrict the term is by no means certain. That the disease does vary more than our writers of even a few years back were aware of, may be admitted, but the variations described in these pages are wider and greater than we have as yet recognised.

The treatment of the subject of alcoholic insanity is well worthy of the large space devoted to it. The author shows none of that sentimental tenderness for the drunkard which has been rife among ourselves. He declares that alcoholic intoxication almost always has its origin in brutal appetites,

and that it is one of the passions, one of the vices of greatest tenacity.

If we may judge from some of the author's remarks, and still more from some of his cases, drunkards are incarcerated as lunatics at the Bicêtre and the Salpêtrière who would scarcely be detained in our asylums. The author says:—

"The most common type (of alcoholic insanity), and certainly the most interesting to study, is that of the individuals whose mental state only betrays itself very rarely by an excitement too violent or an incoherence too manifest, and who either maintain themselves in society, or form in asylums a category relatively superior; at least if one takes the reason for the term of comparison, for it is no longer so if one places oneself at the point of view of the moral faculties."

And he comments without reserve upon the calmness, amiability, and obsequiousness with which these drunkards will deceive the medical man, while in reality their conduct is but a little comedy which conceals the most outrageous lying, rascality and immorality. Evidently the little comedies and beautifully constructed but utterly false autobiographies of the habitual drunkard will never succeed in deceiving this astute observer. He draws, however, a most important distinction between two classes of persons who become insane by alcoholic poisoning. In the first, he places those persons who, being of fairly sober habit, and never having had a previous attack of *delirium tremens*, in consequence of great excess committed within a few days, or even after sudden deprivation of alcoholic drink, necessitated by indisposition or penury, have an attack of mental aberration, consecutive or not to an attack of *delirium tremens*.

In the second class he places drunkards by profession, who have had one or more attacks of *delirium tremens*, and who may be regarded as affected by chronic alcoholism. Not one example of this class on leaving hospital after cure of acute delirium but retains, as symptomatic of his chronic state, enfeeblement of memory and intelligence and of moral energy, diminished aptitude for his habitual employment and loss of muscular force. In some of them the delirium ceases after a month or more, and, notwithstanding enfeeblement of mind,

they may be allowed to leave the hospital; while in others the delirium does not cease, and gradually becomes complicated with confirmed dementia (p. 221).

The number of cases of the first class is small compared with those of the second; during the year only four of the former having been admitted into the hospital, against twenty-eight of the latter. It is rare that after a residence of two months in hospital even the chronic cases cannot be set at liberty. They are not really cured, indeed, but with diminished muscular force and moral energy, and very generally with enfeebled memory, they are yet in a state in which it is impossible to detain them longer. So they leave the asylum, carrying with them the tendency to become again insane with great facility and from slight causes, as, for instance, some excess, or some moral trouble, or even after more or less absolute abstinence from food and alcoholic drink.

The remarks of the author on the delirium of insane drunkards are valuable. He does not agree with some other writers in believing that it is always marked by sadness or depression. It is not a systematic, and, so to say, co-ordinated delirium, like that of monomania. It is essentially illogical, superficial, and impresses its mark rather upon the words and facial expression of the patient than upon his conduct. The expressions of unreason are transitory, and the patient makes light of them if they are disputed. In these respects it resembles the delirium of general paralysis, and differs from that of monomania. The patients, however, do not present the self-control and personal satisfaction which prevails among general paralytics, and this constitutes a diagnostic mark of the two kinds of delirium. Some insane drunkards have amnesia and trouble of speech. Their pronunciation is hesitating, stuttering, confused. Sometimes, perhaps, the hesitation of speech depends upon imperfection of memory, an indication of which is afforded by the fact that the written language is just as imperfect as that which is spoken. The conscience is affected, or that which the author calls conscience. It is impossible to make the sufferer comprehend that it is due to his own fault that no one has been willing to employ him because he was a drunkard, or that his wife has left him

for the same reason. Towards the medical attendant these patients are kindly, submissive, and hypocritical, but towards their wives and children they are far from being so amiable, and are generally irritable and often violent. Without appreciating the cause, they become unable, from loss of muscular strength and freedom of action, to earn their former wages, and this is one reason why they fall into the state of wretchedness which is so common to them; a reason which they do not seem to appreciate.

From the manner in which the author discusses and refrains from deciding whether lunatics from drink can properly make a will, and whether they are responsible for offences against the law (p. 253), it would seem that he does not draw any definite line between drunkards who are not really lunatics, and lunatics who have become such from drink. No doubt there are cases in which the transition from one state into the other is not marked by any well-defined boundary. But the two states are essentially distinct, and the author has failed to define them. Here, as elsewhere, when he trusts himself to any argument or observation outside his rôle as physician, the author lays himself unusually open to criticism. For instance, with regard to responsibility of these people, he says:

"De pareils hommes sont assez le malheur de leurs familles pour que la loi soit en droit de prévenir, par la rigueur de la répression, de semblables habitudes chez d'autres; qu'adviendrait-il, au contraire, s'il était notoire que l'alcoolisme exempte de toute pénalité?"

One would have thought that the continuous misery which a drunkard inflicts upon his family is no reason why he should be exempt from the rigour of the law on account of the occasional misery which he may inflict upon others.

We have no space left for further remarks upon this suggestive volume, except for some brief ones upon the most important subject of treatment. In a few paragraphs the author justifies the use of mechanical restraint, but as a counterpoise he displays the old-fashioned, and we fear the out-of-fashioned, faith of a physician in the efficacy of medicinal remedies. The 28th and 29th lectures are a valuable disquisition on the employment of bromide of potassium in nervous diseases, and

the 30th is a still more important one on the treatment of insanity by the muriate of morphia, endermic injection being the mode of administration mostly used and expressly recommended. Highly as we ourselves value opium and its salts in the treatment of mental diseases, we are not a little surprised at the variety of forms of insanity which have been successfully treated by the author with very large doses of morphia. When he first commenced to treat maniacal, melancholic-persecuted and hallucinated cases with the endermic injection of muriate of morphia, he found it often caused intense vomiting, but, under the advice of Roller of Illenau, he disregarded this inconvenience, and increased the doses with the remarkable success of which his pages are the astonishing record. The contra-indication of the morphine treatment is cerebral congestion, and this he attempts to remove beforehand by blisters to the shaven occiput and nape of the neck. Considering the large doses of morphia which he gave and the long time during which they were sometimes administered, he became apprehensive lest the red blood globules should be diminished by the drug, and he made observations which proved to him that this fear was not well founded. Chronic insanity may itself lead to a diminution of the red globules, but the heroic administration of morphia would seem from the author's observations to resist rather than increase this tendency (p. 703).

The list of the forms of insanity for which he uses the morphia treatment seems to include all varieties of mental disease except those which he considers to be dependent upon a congestive state of the brain. In these it may be injurious, and he well remarks, that this distinction of the forms of insanity, which may and of those which may not be properly treated by morphia, depending upon the state of the organs, as regards congestion, shows that "the diagnosis of the anatomical nature of insanity is of the most serious importance."

The author cites his experience to prove that insanity of many years' standing, five or six years even, may be treated successfully by the morphine method, and that forms of insanity hitherto thought incurable, as *folie à double forme et circulaire*, can be cured by it. In this form the dose of muriate of

morphia needs to be from six to eight grains a day. In lypemania with groaning, the high dose of from seven to ten grains a day is, he says, requisite.

It would be presumptuous to criticise the remedy or the posology of an author who gives such abundant and circumstantial evidence of his success; but it will scarcely fail to strike the readers of Dr. Voisin's volume that muriate of morphia has become with him more of a panacea than will be thought consistent with the variability and many-handedness of scientific medicine. We have no doubt that opium is the most useful of all drugs in insanity; and yet we cannot but express some astonishment at the unexampled success of our French confrère. We can, however, strongly recommend his volume to the study of our psychological compatriots. They will perhaps not accept with implicit faith everything which they find in its pages, but they will find it replete with opinions and experiences gathered in a field of observation new to them, and formed in a widely different spirit to the jejune officialism into which English psychiatry is passing. The volume is illustrated by some excellent engravings and by some interesting photographs. It, however, has no Index, and a very inadequate Table of Matters. Why any one should take the trouble to write a valuable book of nearly eight hundred pages, and not take the trouble, or pay some one else a trifle to take the trouble for him, of constructing an Index, is one of those mysteries of authorship for which we have never yet discovered any reasonable explanation.

JOHN CHARLES BUCKNILL.

Les Hystériques; état physique et état mental. Par le Dr. LEGRAND DU SAULLE, Médecin de la Salpêtrière, &c. &c. Paris, J. B. Baillière et fils, 1883.

FROM France we receive another great work on Hysteria. Although this disease is so widely spread, so universally distributed throughout all countries, climates, and conditions of life, it is a significant fact that the most important literature on the subject is practically confined to certain countries. In England, if we except the writings of Brodie, Todd, Conolly,

Laycock, Skey, and a few others, there are comparatively few special treatises devoted to this question. In France, on the contrary, not only do its medical publications teem with original investigations and observations, but nearly all the great and elaborate memoirs on hysteria emanate from that country, as evidenced by the classical works of Tissot, Louyer-Villermay, Georget, Dubois, Brachet, Landouzy, Briquet, Charcot, and a host of others. Reflection on this point alone would seem to suggest that, although this disease is met with in every part of the globe, in certain special countries it attaches to itself peculiar attention and interest on account of its unusual prevalence or severity. That such is the case is evident from the study of the hysteria at home as compared with the same abroad. Just as the affection seems to attack certain groups of individuals and special constitutions, so do we find it more universal and severe in particular races of mankind, invading those particular temperaments and nervous organisations which would seem to be more specially prone to neurotic excitement and instability.

A consideration of the works of French writers on hysteria indicates not only by their number, but by the nature of their contents, that this disease is much more universal in that country than in England; that phenomena of frequent occurrence there, are extremely rare here; and that the malady, attended with comparatively little danger on this side of the Channel, is surrounded on the other with symptoms of the most serious import. This difference in the two cases is due to the peculiar habits, customs, and mode of education of the respective peoples; and it may be that the fundamental distinction of race is the predisposing cause, in which the nervous organisation of the Latin nation is more prone to this form of derangement than that of the more phlegmatic Saxon. However it may be accounted for, the fact remains that in France, hysteria is a more universal and serious disease than it is in England, where, although common, its type is of a milder and more innocuous form. This, of necessity, attracts public attention; the supply of literature on the subject is in proportion to the demand, and hence the number and importance of works on hysteria in the former country as compared with our own.

The most recent book on the subject lies before us, a volume of 625 pages. To give a complete idea of the contents of so voluminous a work would occupy more space than is permitted us; suffice it to say that the result is eminently worthy of the author, of the hospital he represents, and of the great country from which emanates such valuable neurological researches. It essentially consists of an able *résumé* of all the most modern observations and investigations on hysteria, evidently compiled by a writer of large personal experience, and who has deeply studied the subject, as he tells us, for a period of thirty years. It cannot be said that any startling novelties are advanced, and M. du Saulle throughout largely quotes from Briquet, and on the whole endorses the views of that elaborate and statistical investigator. At the same time the tone of the volume is original, in so far as its conclusions are supported by the personal observations and extensive experience of the author. Above all, the literary style of the book is admirable, and leaves little to be desired; it reads like a romance, and reminds us of the late Sir Thomas Watson's poetical narrations, giving even the dry details of unattractive phenomena a charm which can be perused with pleasure as well as profit.

Without attempting to review this work in detail, we shall direct special notice to one or two points of general interest. In discussing the etiology of hysteria, M. du Saulle proves himself to be one of the modern school who believes that the influence of the uterus in the production of that disease has been, and is, greatly exaggerated. The arguments he employs in favour of this view are precisely similar to those which we ventured to advance in a former number of this Journal (April 1879) in referring to the Hystero-neuroses, and which need not again be repeated. It is there maintained that the genital organs have no necessary connection with the causation of hysteria, although, when disordered, they may, in common with diseases of other organs of the body, be the origin of the nervous phenomena. In short, as the author states, hysterical women have been calumniated when it is insisted that the disorder is due to perverted sexual desires or to functional derangements of the genital organs. On the contrary, it is a neurosis due to instability of the general nervous system as a

result of hereditary transmission, or acquired by unfortunate circumstances, or a faulty education. Such a predisposed constitution is subject, as a result of various exciting causes, to develop those phenomena to which we apply the term hysterical, and among others those of the uterus may claim a share, but not a necessary or unique one. It is known that by far the most common exciting causes are those of a mental or emotional character, and that those of a physical nature are comparatively less frequent. Of these, pain and distress in any form may induce or originate the first manifestations of the disorder, and it is not surprising, considering the prevalence of uterine troubles amongst women, that these should be amongst the number. Gynecologists who see much of hysteria combined with sexual disturbance, and little of it under other circumstances, naturally associate the two together, and many believe the one to be a constant factor of the other; but the fact that such is common in special practice is no proof that hysteria, as a disease, is always or even generally the result of uterine derangement; on the contrary, the experience of the general physician indicates that in the majority of cases there is no evidence of disease of the genital organs, and that when this is present the functional derangement is as often the result as the cause of the general nervous disorder.

One chapter of this work is devoted to an interesting *résumé* of the most recent observations on Hypnotism as occurring in hysterical persons. The author more particularly refers to the researches of his colleague, M. Charcot, at the Salpêtrière. This condition, he explains, may at least assume three different forms in the same subject—the lethargic, the somnambulistic, and the cataleptic—each of which may be either spontaneously or artificially produced, and each of which may be readily transformed into the other. In the first, the patient is in a profound sleep, the senses are blunted, the reflexes are increased, and the muscles and nerves are in a high state of hyper-excitability. In the second, the intellect and senses are acutely sensitive, the reflexes are normal, there is no special irritability of the neuro-muscular system, and the patient readily obeys suggestions from without. In the third, the patient's limbs remain in any position in which they are

placed, the condition otherwise being as in sleep. These, and numerous other complicated and interesting phenomena, are described as occurring in cases of grave hysteria during the hypnotic condition; to appreciate the details of which, the reader should consult the work under consideration, as well as the researches, more especially, of M. Charcot. Such instances of hypnotism, if they exist, must be very rare in this country. Braid and others have recorded cases of somewhat a similar nature, but from all accounts they must be much more frequent amongst our neighbours on the other side of the Channel. The present writer, when in Paris some years ago, saw at the same time in the Salpêtrière a large number of severe cases of hystero-epilepsy, some of whom exhibited the most astounding phenomena under the hypnotic condition. Although he has had ample opportunity in connection with metropolitan hospital practice, and has been on the look-out for such cases for years, he has never met with a single instance of this remarkable form of the disease, and therefore has had no opportunity of testing the effects of hypnotism.

The most novel and perhaps the most important section of this work are the chapters devoted to the medico-legal aspects of hysteria. The author points out that in Paris alone there are no less than fifty thousand persons suffering from the disease who are intimately mixed up in society and in all domestic concerns. That amongst these should arise infractions against the law is not surprising, and, as a matter of fact, the hysterical subject is frequently involved in judicial proceedings. Are such persons responsible agents in the commission of misdemeanour or crime? Such a problem is obviously of the highest difficulty and importance. The author enters fully into the subject, indicating that while many hysterics in offences against the law may be perfectly responsible for their acts, there are others whom it would be a miscarriage of justice to look upon as criminals, as in a certain number of cases the mental faculties are as profoundly disturbed as in serious forms of insanity—of which, indeed, this aberration is only one of the forms. The various states of mind and abnormalities of intellect are detailed, and the different kinds of misconduct and crime fully described, and a

series of typical and interesting cases are given in illustration of the writer's views. M. du Saulle thus studies a question of great practical utility, and one which has been much neglected. He not only investigates the disease itself, and those whom it attacks, but he follows them into society, in its varied relations of civil and domestic life, and into the multiple and complicated conditions demanded by social existence. He looks upon the affection not as a mixture of fanatical caprice or wilful misconduct, but as a serious material complaint, which should be studied on the basis of other corporal diseases, not by appealing to the imagination, but on the dry measurement of facts.

There are many questions considered in M. du Saulle's book to which the space at our disposal does not even permit us to refer. It is sufficient to say that we can strongly recommend the work to the careful consideration of our readers. If it is not profound, elaborately statistical, and bristling with original facts, it is none the less agreeable and profitable reading, and, by its fresh and graphic literary style, pleasantly initiates the English student into the details, from a French point of view, of a disease of which he sees comparatively little in his own country.

A. Hughes Bennett.

La Pellagra nella Provincia dell' Umbria. Del Dott. Roberto Adriani. Perugia, 1880.

In a pamphlet of 78 pages, Dr. Adriani gives us the result of his studies upon pellagra and pellagrous insanity in the Province of Umbrià. This disease, so interesting to the pathologist, is never met with in the British Isles, hence we should have liked if instead of enumerating the bare symptoms the author had given a complete sketch of the course and sequence of the malady. The picture actually presented by the sufferers is, he tells us, very distinctive, though difficult to describe. Pellagra is characterised by muscular weakness, accompanied with pain, anæmia, emaciation, burning in the stomach, colliquative diarrhœa, a livid redness in the face and

hands, a staring and fearful look in the eye, a tottering gait, and a general air of squalor and misery. The loss of strength is gradual, and the nervous symptoms seem to come on at a later period of the disease, consisting in spasms, mental anxiety and discouragement, passing into apathy and stupor. If the loss of strength be not too profound, the patients very often recover during the winter; but the disease is apt to return in the spring. According to Dr. Adriani, about 5·5 per cent. of those affected with pellagra become insane. Some physicians state the proportion as high as 11 per cent. The mental derangement generally takes the form of melancholia, often with great anxiety about religion, and fears of damnation. Not unfrequently there is a tendency to suicide. Sometimes they are troubled with hallucinations; the memory is impaired, the understanding confused, and the speech extravagant. In some cases the appetite is voracious; in others they neither eat, speak, nor sleep. If well cared for and properly fed, they are sent out of the hospital or the asylum with their health restored; but if they return to their old diet, they become worse than ever, till at last they fall into a condition of hopeless dementia.

Pellagra has been in Italy for above a century. It is most frequent in Lombardy and Venetia. On the west it has invaded Piedmont; and on the south it has entered Emilia, Tuscany, Umbria, the Marches, and the Romagna. Towards the south of these provinces it becomes rarer, till it disappears at the confines of the Abruzzi. Pellagra was first seen in 1840; the earliest cases appeared about the Thrasimene lake. Year by year it becomes more common, It was at first confounded with paludal malaria. From 1847 to 1879 about a thousand cases have been observed in Umbria. In some cases the mental symptoms are slight and transitory. About a third of those who become insane are treated in their own homes. From 1854 to 1880 there have been 306 admitted into the Asylum of Perugia from pellagrous insanity.

Dr. Adriani gives the population of Umbria at 549,203 inhabitants, of which 273,292 are dwellers in the country, and 245,767 are engaged in agriculture. He calculates that there are 2·8 pellagrous for every thousand of the general popula-

tion; 5·7 for every thousand of the rural population; and 6·3 for the agricultural population. One reason why the disease does not excite more attention is that its ravages fall principally upon the poorest of the country people, who suffer in silence.

Dr. Adriani feels himself constrained to combat the views of superficial theorists, who attribute pellagra to an insufficient diet, to unhealthy surroundings, overwork, want of flesh-meat, or insolation. Were the disease owing to any of these causes, severally or jointly, assuredly its distribution would be much more extensive. Like goître, cretinism, or malarious fever, it must be owing to a specific cause. The arguments adduced by Dr. Adriani, that pellagra is the result of an altered or degenerate condition of the maize which forms the staple food of the peasantry, seems to be of irresistible force.

Pellagra is observed only where maize is an article of food, and it appears in localities where it was previously unknown after maize comes into use, quite independently of the condition of the air, of the nature of the soil, of the quantity and quality of the water, and of the healthiness of the houses. The disease, moreover, becomes aggravated and diffused in proportion as the consumption of Indian corn is increased and extended. In those years in which the corn and rye crop was scanty, and maize entered more largely into the dietary of the peasantry, pellagra became more frequent; whereas it disappeared where the maize crop failed, as in the province of Brescia in 1816 and 1817. It has been asserted by Henry Gintrac, in the article "Pellagra," in the 'Nouveau Dictionnaire de Médecine et de Chirurgie Pratique,' published in 1878, that the disease has appeared in cases where maize was never used for food. Dr. Adriani has examined some of these instances, and finds them incorrectly reported. In one case a woman had given up the use of Indian corn by the advice of her medical attendant after she had fallen ill. Calling in another physician, she had told him that she was not using any maize, on which he had assumed that the disease had been produced without the use of that grain.

In another case which came under the direct observation of Dr. Adriani, the mother of the family had herself lived upon

maize, which she had obtained at a low price from the miller, and had given up her share of better food to her children. The doctor, not knowing of the self-denial of the poor woman, published the case as one of sporadic pellagra coming on without the use of maize.

Small properties seem to be common in Umbria. Where the proprietors do not cultivate their own land, they give it to metayers, who divide the produce with the proprietor, generally in equal halves, the proprietor furnishing the seed. This answers better than where the land is given to small farmers, who get into debt and exhaust the soil.

The labourers who work for the metayers are very poor. Maize forms a proportion varying from two-thirds to four-fifths of the dietary of the agricultural class. Besides this, they eat beans with rice, potatoes, and cheese in small quantities; oil and salt are the condiments used. They generally drink light wine, but seldom eat flesh. The poorest classes live principally upon maize, half cooked, with a little salt. The kind of Indian corn mostly seen is the Zea mais vulgaris æstiva; more rarely the Vulgaris autumna. The culture of maize is always extending, hence they are not so careful in choosing situations where it may be expected to ripen perfectly. Italy consumes more maize than it produces, so there is a considerable importation from Albania and the Danubian Provinces. In a village in Poland pellagra once appeared, from the inhabitants using maize brought from the Danubian Provinces. It would appear that often there is not heat enough in the summer of Northern Italy fully to ripen the grain. The immature and damp corn is liable to degenerate. This tendency is often increased by the manner in which it is stored. In Corfu an epidemic of pellagra followed a cold and wet summer. Balardini found that he could produce symptoms similar to those of pellagra by giving spoiled maize to animals as well as to human beings. Lombroso, by administering for a long time small doses of a tincture of degenerated maize, containing a red oil of a bitter taste, produced some of the symptoms of the disease—such as voracious appetite, with nausea and burning of the stomach, heat and pruritus of the limbs, diarrhœa, emaciation, and weakness. Through the

researches of Lombroso, which were at a later time associated with those of Dupré, and of Pesaro and Ebra of Milan, an oily resin and an azotized alkaloid, called pellagrozeina, were discovered. This substance resembles strychnine in some of its chemical properties. Pellagrozeina is said to be of use in some diseases of the skin. Given to chickens, it produced diarrhœa, paralysis of the limbs, tonic convulsions, and death. With grown-up cocks it produced immobility, diarrhœa, and paresis of the lungs. Given to dogs, it was found to cause somnolence, diarrhœa, thirst, refusal of food, and diminution of weight. In a man who had swallowed some pellagrozeina there followed confusion in the head, nausea, diarrhœa, and disgust of food; in another man there was a sense of weight in the lower abdominal region, burning in the throat, loss of appetite, pruritus, and melancholy. The experiments with the drug were pushed so far as to produce death in animals. It was found that the effects varied when the pellagrozeina was obtained from maize. The spinal cords examined showed indications of diffused granular myelitis, with a characteristic softening of the white substance, and in some cases of the grey matter. This softening sometimes occupied the whole cord; in other cases it only implicated the lower portion. There was also fatty degeneration of the sympathetic nerves. Dr. Adriani points out that pellagra has already had a serious effect upon the general health of the rural population of Umbria. It has brought about a notable diminution in their physical force and intelligence, has increased the number of the insane and suicidal, and raised the annual mortality. He discusses in a comprehensive manner the question how to prevent and remedy the disease; but for these and other interesting passages we must refer to the pamphlet itself.

W. W. Ireland.

Clinical Cases.

NOTES ON A CASE OF CEREBRAL TUMOUR.

BY A. HUGHES BENNETT, M.D.

Physician to the Hospital for Epilepsy and Paralysis, Regent's Park, and Assistant Physician to the Westminster Hospital.

T. K., ætat. 49, Railway Guard.—*Antecedent History.*—The patient says that he has always been a perfectly healthy man, but his wife states that for some time past he has been irritable and depressed, and had deficient memory. There is no history of syphilis, and with the above exceptions he has been considered in robust health, till about two months ago, when for the first time he observed slight weakness of his left leg. This has gradually increased till the present time. Two weeks ago, being in this condition, in the course of a day his left arm became very feeble, which has slowly progressed since, but he has managed to continue his work till three days ago. There has been no pain in the head, nor has he ever suffered from fits, or from cerebral or other abnormal symptoms.

Present condition.—The patient has all the appearance of a man in robust general health, and all his functions, except those to be described, are normal. There is no pain in the head or elsewhere; the special senses and the actions of all the cranial nerves are normal, except that the tongue, when protruded, is pushed very slightly to the left side. There is no optic neuritis. The face is perfectly straight, and the speech is unaffected. The movements of the left upper extremity are greatly impaired, those of the shoulder-joint are very weak, those of the elbow more feeble, and those of the wrist and fingers abolished, so that there is complete wrist-drop and complete immobility of the whole hand. The left arm is colder and bluer than the right. The sensibility is everywhere normal, and there is no wasting of the muscles. The patient can walk, but is very lame with the left leg, which is feeble, and has a tendency to sink under the weight of the body, but there is no rigidity or special gait. When sitting

on a chair the movements of the lower extremity can be performed, but slowly and weakly, and its sensibility is normal. The cutaneous reflexes are as in health, the knee-jerk phenomenon is most marked on the affected side, where there is also evidence of slight ankle-clonus. There is no wasting of the muscles. The electrical reactions are the same on both sides, and normal.

Progress of the Case.—The patient was under observation three weeks in the hospital, under the care of Dr. Sturges, before he died. The entire left side gradually became weaker, and was occasionally attacked with paroxysmal twitchings, which included the side of the face. Slight hesitation of speech was noticed, with a tendency to the repetition of words, but this was very indefinite. Soon after supervened dulness of intellect, depression of spirits, a feeling of numbness of the left leg, and slight twisting of the face towards the right side. These symptoms continued to increase, so that a few days before death he was dull, partly comatose, and very difficult to rouse; the speech was much embarrassed, and he had visual hallucinations, fancying he saw all sorts of things and people in the ward. The paralysis of the left side became complete, he passed all his motions in bed, vomiting ensued, the pupils were strongly contracted, deep coma, and death ended the scene.

Post-mortem examination, (18 hours after death).—Permission was only given to examine the head. The following notes are mainly abstracted from the report of Dr. Hebb. The body is well nourished. The membranes of the brain are healthy, and there are no evidences of meningitis. The blood-vessels are everywhere normal. The convolutions of the brain are flattened on both sides. There is no excess of subarachnoid fluid. On removing the membranes, just anterior to the fissure of Rolando is seen a circular patch the size of a crown, of mottled appearance, and consisting of softened or broken-down grey matter. This occupies nearly the whole breadth of the ascending frontal convolution at its middle, but not extending quite as far back as the fissure of Rolando. It also involves the whole of the posterior end of the middle frontal convolution, and a small portion of the bases of the superior and inferior frontal gyri. On making an incision through this softened area in the line of the ascending frontal convolution from the surface down to the ventricle, it is found that a mass of spheroidal shape, and about the size of a small orange, occupies the centrum ovale of the right hemisphere, its apex constituting the floor of the cortical softening, and its base resting on the roof of the lateral ventricle, into

the cavity of which it projects, without, however, apparently pressing upon the structures in its interior. The tumour is a gelatinous-looking mass, consisting apparently of broken-down nervous structure, and traversed by dilated blood-vessels, and was subsequently ascertained to be a glioma. It is sharply defined from the neighbouring nervous tissues, which appear healthy. The remainder of the brain is normal.

Commentary.—On this case little comment is necessary, the facts being simple and apparent. Beyond the gradual and progressive appearance of the paralysis, there were no symptoms during the life of the patient to indicate tumour of the brain. There was no pain in the head, no marked implication of the cranial nerves, no convulsions or sickness, and no optic neuritis. It would seem as if the lesion originated in the centrum ovale, where, by slow growth, and gradually pushing aside the tissues without destroying them, it attained considerable size without causing symptoms of any kind. Finally, some weeks before death, by extending towards the cortex and involving its structures at the motor centres, or by pressing upon their conducting fibres, paralysis was induced.

On the question of cerebral localisation this case does not throw any positive light, as the tumour was not confined to the grey matter of the cortex alone, but extended largely into the medullary substance below; and although the basal ganglia and structures of the brain generally, elsewhere appeared healthy, it is always difficult to estimate how far in such a case the effects are due to disease of the convolutions, or to pressure on or destruction of the conducting media. The area of degenerated cortical substance did not involve those portions of the convolutions in which are supposed to originate movements of the lower extremity, and only to a very slight extent those presiding over the motion of the arm. During life, however, there was marked paralysis of both these limbs. This, of course, proves nothing against the observations advanced in favour of cerebral localisation, as the tumour extended downwards in the conducting matter, and may have thus interfered with its function in transmitting influences from the parietal cortex to the periphery, without the former being itself directly implicated.

The lesion, however, suggests certain negative conditions which may be noted. The patch of softened grey matter was definite and complete, occupying the area already described. Within this space is what is supposed to be the oculo-motor centre, which on this side must have been in great part destroyed. During life it was certain that the patient displayed no abnormalities in connection with the movements of

the eyeballs, the pupils, or the eyelids. It may be, as Ferrier suggests, that the disease proceeding slowly, and one side being healthy, the bilateral association of the oculo-motor nuclei may compensate for disease on the other.

The area of softening also involved what is stated to be the centre for facial movements. It was, however, particularly noted that until a few days before death the face of the patient was in no way affected. It is true that near the end a deviation was observed, but this was very slight.

These points are here noted as matters of fact, and not with the view of throwing doubt on the question of cerebral localisation, for, as already stated, the nature and distribution of the morbid lesion utterly precludes any accurate conclusions on this subject. In this case we are even uncertain of the exact limits of the destructive process in the grey matter, and of its duration in point of time, not to speak of the complications arising from the presence of a large tumour in the encephalon.

The diseased mass must have been in close proximity to the right island of Reil, and being on that side probably accounts for the absence of any defect of speech. Towards the close, slight difficulty of articulation was noticed, but there was no true aphasia.

Finally, it may be noted that in this case of cortical lesion there were no convulsive attacks. A few days before death, occasional twitchings of the left arm, leg, and side of the face were observed, but these were very slight.

LEFT HEMIPLEGIA AND LEFT-SIDED DEAFNESS AFTER WOUND OF BRAIN THROUGH RIGHT ORBIT. PROBABLE LESION OF INTERNAL CAPSULE AND FERRIER'S AUDITORY CENTRE.

BY H. DONKIN.

THE following case of accidental "vivisection" in the human subject is placed on record for its clinical and physiological interest.

R. Putt, a boy of twelve years old, was carried into the East London Hospital for Children at Shadwell, at 5 P.M. on Feb. 16th, 1882, in a sensible, but very drowsy state, with the history of having had the steel rib of an umbrella "run into his eye." It appeared that in playing with his schoolfellows he had been shut into a room alone, and upon this, applied his right eye to the keyhole to see what was going on outside. One of the boys, not knowing that the eye was on the other side, thrust forcibly through the keyhole a detached steel umbrella-rib, which, according to R. Putt's positive statement after his recovery, entered the outer corner of his eye. He felt giddy, fell down, and then, while on the floor, pulled out the spike. He cannot say how far it went in. Nothing more was remembered by him till he was brought to the hospital about twenty minutes or half an hour after the accident.

I saw the boy immediately after his being taken to the Casualty Room. He answered when spoken to, but had considerable difficulty in articulation. His left arm seemed quite powerless, falling heavily down when lifted, and he could scarcely move his left leg. The mouth was markedly drawn up towards the right side, and the tongue, protruded with some difficulty, pointed to the left. The right eye was invisible, the tissues around being much swollen and discoloured by extravasation.

Half an hour after admission to the ward, the boy's condition was noted by Mr. J. Scott Battams, the Resident Medical Officer, as follows:—

"Boy sensible but drowsy: has just vomited. Speaks in a

drunken spluttering fashion: says he felt giddy after the accident and could not stand. Has pain in the occipital region. Considerable loss of power in left leg: resists flexion and extension very feebly: can just raise the leg from the bed. Left arm weaker than leg: cannot squeeze with the left hand at all. No pain or affection of sensation in paralysed limbs. Sole and patella reflexes present: cremaster reflex not elicited on left side.

"Right eyelids much swollen, and opened with difficulty only enough to expose a small part of the eyeball, which is tense and seems pushed forward. No distinct wound to be made out.

"Cannot hear watch in left ear: hearing perfect on the right side. Left side of face paralysed: cannot wrinkle forehead on left side: right angle of mouth drawn up. Sensation normal. No affection of left eye. Tongue, protruded, points to left. No rigidity or spasm of limbs. Pulse 100, irregular in force and frequency. T. 98."

At 10 P.M. (same night) the boy was noted to be lying on his left side: he would not lie on right side or back. He had vomited twice. He was very drowsy, but quite conscious. He was said by the sister to have had some convulsive movements of the *right* arm. When the right hand is held there are some convulsive movements of the fingers.

The next morning (17th) the following note was taken:—"Slept well, not sick since 2 A.M. Slept with hands at back of head. Cheeks flushed, more on left side. Is quite sensible and speaks plainly. Still deaf in left ear. Tongue still points to left. Now only complains of pain in left occipital region. Can now raise the left arm from the bed: and can resist flexion and extension of the leg better. Right side of face still drawn up. No loss of sensation on left side, but the left hand and fingers are said to feel as if wrinkled."

18*th*.—"Decidedly better. No pain. Talks more plainly. Power in limbs increased: coarse and fine movements of left hand and fingers well performed. Still facial and lingual paralysis, though less marked. Less swelling of right eyelid: can see indistinctly with right eye. Vision in left eye normal. Pulse 80, regular. Last night the pulse was intermittent. *Cannot hear on the left side a watch placed close to the ear, or on the skull.*"

21*st*.—"Boy has gone on improving. No headache, tongue protruded straight. Facial palsy much less marked. There is considerable power in the left limbs. No twitchings."

23*rd*.—"All paralytic symptoms have disappeared from the left side, including the face and tongue. The right eyeball is

now, owing to subsidence of the swelling, completely uncovered. It can be moved only in the upward direction, and but slightly. In all other directions it remains motionless as the left moves. He can see with the right eye, though indistinctly."

After this date no fresh symptom showed itself: the boy appeared perfectly well. Gradually the movements of the ocular muscles of the right side returned, and on March 20 the paralysis was apparently confined to the external rectus muscle; all other movements being well performed. The right pupil is slightly larger than the left, and acts more to light.

On admission, the boy's temperature was 98. For the first three days after the accident it varied between 99 and 100, and was afterwards normal.

On May 8, after being at the convalescent home, the boy was seen again, presenting no abnormal symptom other than a slight squint, owing to a small degree of loss of power in turning the right eyeball outwards. He made no complaint at this time of seeing double, but was found to do so on June 6, when he was re-admitted for a short time, owing to an alleged slight weakness of his left leg. The fundus of the right eye showed nothing abnormal. He complained of not being able to read for any length of time. Sees double, unless he closes the right eye, and complains of pain in it when he tries to read. The false image is to the right, and a little above the true one. Cannot hear so well with the left as with the right ear, but there is no complaint of deafness. *Periosteal hearing on both sides perfect.* There is no discoverable mark of any wound in the structures of the right eye, nor was such at any time made out. There is no evidence of any weakness of the left leg.

Remarks.—This case of injury to the brain, producing motor and probably auditory paralysis of the opposite side, is of great physiological interest, owing to the apparently strict limitation of the injury. Such limitation is shown by the definite nature of the functional disturbance produced, and the recovery of the patient without marked constitutional symptoms. It is, moreover, rendered probable by the character of the instrument which penetrated the orbit, an ordinary umbrella-rib, having at the end, which in the complete umbrella is connected with the central part of the frame, a tolerably sharp chisel-shaped edge.

The symptoms to be accounted for are mainly these :—

1. Left hemiplegia of the ordinary type, with rapid recovery.
2. Probable nerve-deafness on left side, with recovery.

3. The paralysis of the extrinsic muscles of the right eyeball.

1. The hemiplegia being of the ordinary pattern implies, with the greatest probability, not a cortical lesion, but some injury to the motor tract in the internal capsule of the right side.

2. The deafness (of which there was no history or suspicion previously to the accident) being an *isolated* sensory symptom, points probably either to a right-sided cortical or left-sided nerve lesion. On the justifiable assumption of the impossibility of a multiple lesion, not to mention the serious symptoms which must have resulted from the passage of the instrument to the base of the brain on the left side, the theory of injury to the auditory nerve may be dismissed, and a cortical lesion inferred.

With these inferences the facts of the case remarkably coincide. The symptoms are explicable by the supposition of an injury to the internal capsule and to the superior temporo-sphenoidal convolution (or Ferrier's auditory centre) on the right side.

The instrument above described, under the conditions alluded to, might easily penetrate the thin bone of the orbit, and having once entered the brain would have no obstacle to its further progress. The probable course of the instrument was demonstrated to me by Dr. Ferrier, who kindly studied at my request the notes of the case, and showed me by means of a skull, with surface markings corresponding to the convolutions beneath, that the spike having pierced the orbit on the outer side, and passing nearly straight backwards in a horizontal plane, might easily have traversed the region of the island of Reil, injuring the internal capsule, and then found its way into the superior temporo-sphenoidal convolution. Thus, then, the two salient symptoms of *hemiplegia* and *deafness* are satisfactorily accounted for. It will be observed that the temporary nature of the symptoms is in accord with the probable character of the wound caused by the instrument in question. Such a wound would be in itself slight; the symptoms being probably caused by the pressure of effused blood in the track of the instrument.

3. The paralysis of the extrinsic muscles of the right eyeball, which gradually disappeared with the exception of that of the external rectus, is best explained by a local injury done by the passage of the spike through the orbit, setting up a considerable degree of extravasation, and probably some inflammation of the structures in that region. The boy very clearly stated

that he was sure the spike ran into the outer corner of his eye —a fact which falls in with the more permanent injury done to the nerve of the external rectus.

P.S.—I have lately (Nov. 20, 1882) seen the boy again. I find that he has now a discharge from his left ear, which interferes with his hearing by the meatus. But his hearing is equally acute on both sides when tested by a watch placed on the skull. This discharge from his ear has taken place from time to time for several years, according to his mother's statement. While in the hospital the boy had no discharge, and the history taken at that time did not reveal any previous deafness, neither the mother nor the boy having noticed it, as appeared on inquiry.

This fact does not negative the probability of the deafness observed after the accident being caused by an injury to the auditory centre. For there was at that time a complete inability to hear a watch placed on the skull on the left side; while the present condition shows no impairment of the function of the auditory nerve. Mr. Clutton, of St. Thomas's Hospital, has kindly examined the case for me, and reports normality of the nervous apparatus on both sides, with tympanic inflammation on left and Eustachean obstruction on right side. He considers this condition to be of long standing.

COMPOUND FRACTURE OF SKULL AND ABSCESS OF FRONTAL LOBES.

BY J. M'CARTHY, F.R.C.S., ETC.

T. W., a boy aged 6 years, was admitted into the London Hospital on March 18th, 1882, with a compound fracture of the skull, caused by his having been run over by a hansom cab. There was a lacerated wound on the left temple, a little above the zygoma, communicating with the fracture, which could be traced under the skin transversely across the forehead, a little above the supraciliary ridge, forming a prominent line with a considerable depression above. Both upper eyelids were swollen with extravasated blood, and there was a small but rapidly increasing hæmorrhage beneath the left ocular conjunctiva. The pupils were active, and of equal size. The nasal bones were displaced forwards, the right in advance of the left. The child was dull and fretful, but when roused, answered intelligently, and there was no loss of motion or sensation. He had bled copiously from the nose, and vomited coffee-ground fluid. I cut down upon the apparent depression with the intention of elevating it, but found that the deformity was produced by the lower part of the frontal bone with the nasal bones having been forced forwards in advance of the rest of the skull. It thus became evident that the fracture had traversed not only the vertical plate of the frontal bone, but also both orbital plates and the cribriform plate of the ethmoid. Any effort to replace the bones in position led to such abundant escape of brain-substance that the attempt had to be desisted from. He was sent back to bed, the wounds having been dressed with lead lotion, and an ice-bag was applied to the vertex. The next morning he was drowsy, but when roused could answer intelligently, and recognised his mother. He took fluid nourishment fairly well.

During the next few days he continued much the same, but the temperature gradually rose. On the fifth day it was a little over 104° Fahr. and inunction of mercurial ointment was ordered. His gums speedily became spongy, and on the eighth day the temperature had fallen to 100° Fahr., and

continued about the same, with slight variations, for some time, gradually falling to normal. Both upper eyelids suppurated, and there was a profuse discharge of matter from them. He, however, improved in general condition, and his appetite was good, but when disturbed in any way uttered a peculiarly shrill and piercing cry.

About the sense of smell no satisfactory evidence could be obtained. All the wounds healed well and rapidly, and by April the 19th he had become so lively as to necessitate his being fastened in bed, to prevent his getting up and running about the ward. On May the 20th, slight apparent ptosis of the left upper eyelid was noticed, but on further examination it was evidently the result of the roof of the orbit becoming gradually depressed so as to interfere with the upward movement of the eyelid. The optic discs were examined from time to time, but were always normal. He was allowed to get up, and was now always playing about the ward with the other children. The nurse reported that he was sometimes obstinate, and got into a temper, but was of a lively disposition, and always had a good appetite.

May the 22nd there was renewed suppuration in the left eyelid, and a rise of temperature, which subsided again after the matter had discharged. Nothing worthy of record occurred until the evening of June the 7th, when he was seized with convulsions, and the right side of the face was especially affected. After this he again rallied, but at midnight, on the 10th, had another severe fit, in which the left side of his body was chiefly affected. Bromide of potassium was ordered. During the next week he was restless at night, had twitchings of muscles on both sides of the body, and complained of pain in the right hip, for which no cause could be discovered. On July 1st the left eye was noticed to be prominent, and a small swelling over the left frontal eminence appeared. At this part the fractured bones were found to be again separated by some growth from within the cranium pressing forwards. During the next ten days this increased, and the swelling was punctured, when some grumous matter escaped. The child now lay perfectly quiet, unless when disturbed, when he could be roused to partial consciousness. There was incontinence of urine and fæces. The stupor gradually increased until his death on the 22nd of July.

At the *post-mortem* examination the anterior fossa of the cranium was found to be fractured, and a disintegrated brain-substance from the under part of the left cerebral hemisphere protruded into the left orbit and the nasal cavity. The left frontal lobe was a diffluent mass. The right adhered firmly

to the membranes, and on separating it the grey matter shelled off, remaining adherent to the membranes, while the white substance gave very distinct fluctuation. On cutting into it, about three ounces of thick pus escaped, but the walls of the cavity were so rigid that they would not collapse.

The case is interesting, partly from the unusual nature of the fracture and resulting deformity, but chiefly from the severe injury which the anterior part of the brain must have sustained at the time of the accident without there being any symptoms to indicate it. With the exception of the sense of smell, his other faculties were normal until within about ten days of his death, and for about six weeks of his hospital life he was active, merry, and extremely intelligent for his years.

The temperature-charts exhibited occasional elevations, particularly in March 23, May 2, May 23, June 13 and 16, which were accounted for at the time by the occurrence of suppuration or diarrhœa.

HYSTERICAL MANIA: ATHEROMATOUS DISEASE OF LEFT INTERNAL CAROTID: THROMBOSIS OF ARTERIES AT BASE OF BRAIN, WITH CONSEQUENT SOFTENING OF CEREBRAL SUBSTANCE.

BY W. B. KESTEVEN, M.D.

THE following case presents points of interest, both psychologically and pathologically.

On the 30th of September last, I was requested to undertake the care of a lady who was said to be suffering from a severe form of hysteria. The patient came to me in the afternoon of that day, was perfectly collected and cheerful, entered into conversation in the intelligent and self-possessed manner of an educated lady. She joined our family party at tea, and in the course of the evening sat down beside a whist table, to watch the game, as she herself said. In about half an hour she suddenly left the room and retired to her bedroom, where, on being followed, within a very short time, she was found almost in a state of nudity, complaining that her bowels would not act, and endeavouring to evacuate the rectum with her fingers. She talked incoherently, was incessantly moving about; so restless that it was impossible to keep her in bed by any persuasion, although at the same time declaring herself paralysed; she passed her evacuations under her. On the following morning she became quieter, kept to her bed the greater part of the day, but was still incoherent. She took no food except by strong persuasion. On the morning of the third day, while standing up to be dressed she suddenly fell to the ground, but got up again, all the while protesting that she had lost the use of her legs. She seated herself on the floor in the corner of the room, so as to support herself on either hand by the angles of the wall. She was cheerful, even loquacious, for a few hours.

It became necessary to remove her to an asylum, which was effected by carrying her, as it appeared that she would not, or could not, stand. The sequel shows that she could not. As the case, however, had previously been regarded as one of hysteria, her want of muscular power was doubted. Dr. Wright, of

Northumberland House, Finsbury Park, under whose care she was placed on Oct. 2nd, has obliged me by notes of her condition whilst under his observation. On admission, she was semi-comatose, and in a state of profuse perspiration. She did not move or speak. She would half-open her eyelids and gaze furtively around; her eyes were turned to the left. She seemed to be, to a great extent, aware of what was going on. She gave the impression that she was simulating paralysis and stertorous breathing. Her right side was "limp," but no marked difference from the state of the left side. No reflex action elicited on soles of the feet. Before she died there was a distinctly paralytic state of the left cheek and ptosis of the left eye. Her temperature rose to 103°, and her pulse to 130 during the day of her death, Oct. 4th. Dr. Wright has kindly furnished me with a copy of Dr. Goodhart's report of the macroscopic examination of the brain.

INSPECTION MADE 4 P.M., OCTOBER 5, 1882.

Cranial Bones.—Thin.

Dura Mater and Sinuses.—Healthy, except that dura mater was unduly adherent to skull.

Arachnoid and Pia Mater.—All looked quite healthy, but when stripped from the grey matter beneath on the left side the surface beneath was left ragged and soft; the colour being unduly yellow, from the intermixture of blood pigment.

The *vessels* were all healthy except one, and that the left internal carotid. This vessel was obviously plugged by firm substance, and on making a transverse section of this part and examining it closely, it became evident that the coats of the vessel were extensively diseased here; that atheromatous thickening had occurred, leading to considerable diminution of the calibre of the vessel. This had, of course, been going on for some time, probably many months. More recently, perhaps in the last two or three weeks, perhaps even more lately than this, the diminished channel had become completely closed by fresh clots being deposited upon the roughened arterial wall.

The transverse section of the artery thus showed an outer thick yellow zone, or partial zone, and an inner claret-coloured part.

Looked at from above, the left hemisphere was markedly fuller than the right, and bulged out, more particularly in the lower part of the anterior, central, or ascending frontal convolution. It was in this part that the membranes, when stripped, left a ragged surface behind. Making sections of this hemi-

sphere, it was found that the central cortex about this convolution—that is to say, it and the convolutions adjacent—was extensively swollen, so as to have lost its outline of demarcation from the white matter beneath; it was ecchymosed in many places, minutely vascular in many more, and everywhere had lost its consistence when compared with the opposite hemisphere and the sounder parts of this one.

The lenticular nucleus, the corpus striatum, had suffered in the same way—bloated-looking and pulpy.

It was particularly noticed, and this has an important bearing on the symptoms, that except in the superficial layers of the grey matter, which were beginning to disintegrate, there was as yet no evident solution of continuity of the brain-fibres, and thus there had been no marked paralysis.

The ventricles and other parts of brain all looked healthy.

To the preceding I would add the results of my microscopical examination of portions of the convolutions taken from near the seat of the softened substance. These may be summed up as disintegration of structure by softening—atrophy of nerve-cells, dilatation of the minute vessels, and extensive miliary degeneration—conditions, all of which are indicative of longer-standing disease than would appear from the history of the case. The patient had always been regarded as hysterical. In the month of June last she had a transient attack of hemiplegia. After this she experienced paroxysms of hysteria, bordering upon mania; she complained of tingling sensations in various parts of her body.

This case is worthy of note on account of the difficulty of diagnosis in its early stages, and the contrast presented between its later symptoms, compared with the extent of pathological lesions revealed after death.

Abstracts of British and Foreign Journals.

Stenger on Cerebral Affections of Sight in General Paralysis.— Stenger (*Archiv f. Psych.*, Bd. xiii. p. 218) reports five cases illustrative of a peculiar affection of sight which Fürstner, several years ago, described as occurring in the course of general paralysis of the insane. The symptoms are generally observed after the epileptiform seizures that occur in this disease. The patient can see objects and follows them with his eyes, but he has no ideas about them; they do not recall the associations they used to do, or call forth the same actions. For example, he shows no signs of fear if a burning stick is suddenly thrust before his face, perhaps he will try and catch hold of it. Though he sees an obstacle in his path, he will continue in his course till he stumbles over it. If a glass of wine is held before him, it does not seem to occur to him that it is for drinking; it is only after it has been pressed against his lips that he shows his appreciation of it, and drinks it. The patient sees, but he does not understand; just as a man cerebrally deaf hears, but does not understand. This condition lasts for a variable time. In the first case it lasted about ten days, and then rapidly disappeared, and sight was normal for three or four weeks, when another attack came on, which was in turn recovered from. After a series of such attacks and recoveries, during which the dementia and paralysis gradually increased, the patient died. In two cases there was absolute blindness for several days, which was succeeded by the condition of mental blindness described above.

It will thus be seen that there are two distinct conditions, one of absolute blindness or cerebral amaurosis, or, as it is called by Munk, cortical blindness, *rindenblindheit*; the other of partial blindness, psychical or mental blindness, the *seelenblindheit* of Munk.

The cases that Stenger has observed differ in some respects from those described by Fürstner. In Fürstner's cases, only one eye was affected; in Stenger's, with one exception, both eyes were involved. Stenger always found paralysis and dilatation of the pupil; while Fürstner states that the contractility of the pupil is retained.

Post-mortem examination showed that the symptoms were due to disease of the cortex cerebri; but the lesions were too diffuse to allow of any conclusion as to the localisation of the sense of sight.

Stenger reports several cases of general paralysis in which he has observed hemianopsia, without the peculiar symptoms of impaired vision just described. A man had an apoplectic seizure, and lost power over his left face and extremities. Three days afterwards, by which time he had regained consciousness and intelligence, there was found left hemianopsia and hemianæsthesia. The hemianopsia continued for about a fortnight, and then gradually disappeared, but returned in a few months after a fresh paralytic attack.

In another case, left hemianopsia appeared after a paralytic attack affecting the left side. Three months afterwards, right hemianopsia developed, and was followed by convulsions of the right arm and face. The patient was now quite blind, and remained so till death, which happened four weeks afterwards. During the early part of this period he was able to converse rationally, and the other special senses were normal. On two occasions he had hallucinations of sight. On post-mortem examination, in addition to other lesions, the occipital lobes were found to be much atrophied, and the pia mater injected and firmly adherent to the softened cortex.

Zacher on some peculiar forms of General Paralysis.—Zacher (*Archiv f. Psych.*, Bd. xiii. p. 155) relates a case in which in addition to the ordinary symptoms of general paralysis, there were the characteristic symptoms of spastic spinal paralysis. The only symptom, indeed, in which the case differed from a typical case of spastic spinal disease was the presence of a general hyperæsthesia. In similar cases that have been described by Claus, Schultze, and others, sclerosis of the lateral columns of the cord has been discovered. But in this case no pathological change was found, either in the lateral columns or in any other part of the cord. Zacher attributes the spastic symptoms to lesion of the cortex cerebri, more particularly that part of it from which the pyramidal strands spring. He suggests that a differential diagnosis between the cases of general paralysis in which there is sclerosis of the lateral columns of the cord, and those in which there is no sclerosis, may be founded on the absence in the former cases of decided sensory symptoms and well-marked contractures.

Zacher reports another case which presented the symptoms and the post-mortem appearances both of multiple cerebro-spinal sclerosis and general paralysis. Instances of a similar combination have been recorded by Claus and Schultze (see 'BRAIN,' Vol. II. p. 142; Vol. IV. p. 268).

Ganser on the Optic Nerve.—Ganser, as the result of experiments on cats (*Archiv f. Psych.*, Bd. xiii. p. 341), comes to the conclusion that the non-decussating fibres of the optic tract form a distinct bundle, which runs along the lateral border of the optic chiasma and the optic nerve.

In confirmation of this view, the author cites the case of an epileptic, in whose brain a thin band of fibres was seen to separate itself from the right optic tract a little in front of the corpora geniculata, pass forwards on the ventral surface of the tract to the lateral border of the chiasma and nerve, and afterwards become incorporated with the nerve. This view is in direct opposition to that of Kellermann, which denies the existence of a fasciculus lateralis, and asserts that all the fibres intermingle in the chiasma. Ganser analyses the case upon which Kellermann's theory is based, and declares it unsatisfactory and indecisive.

The non-decussating fibres are distributed to the temporal half of the retina, the decussating to the nasal half and to parts of the temporal half. The area centralis seems to be supplied by both kinds of fibres. After destruction of the decussating fibres of the optic chiasma, there is atrophy of the nerve-fibre layer, and of the ganglion-cell layer in the nasal portion of each retina.

Removal of the posterior parts of the left cerebral hemispheres of new-born cats caused homonymous right hemianopsia, and on the animals being killed nine months afterwards, the left optic tracts were found atrophied, and the atrophy was less extensive in the left optic nerve than in the right, showing that the decussating fibres are developed more largely than the non-decussating. The nerve-fibre layers were thinner on the left side of each retina; in other words, there was a homonymous left-sided atrophy of the layers.

Ganser's paper concludes with some observations on the anatomy of the corpus bigeminum anterius.

Meyer on a Case of Hæmorrhage into the Pons with Secondary Degeneration of the Fillet.—A man, aged 48 (*Archiv f. Psych.*, Bd. xiii. p. 63), after a slight apoplectic seizure

was found to have complete paralysis of the right facial and abducens nerves, with conjugate paralysis of the left rectus internus, anæsthesia of the left half of the face, trunk, and extremities (least marked in the face), hyperæsthesia of the right half of the face, and paresis of the left limbs. The paresis soon disappeared, leaving only ataxia of the left arm. Headache and vertigo were prominent symptoms in the case.

On post-mortem examination a hæmorrhagic focus was found in the tegmentum in the right half of the pons. The lesion was confined to the lower two-thirds of the tegmentum, and involved the following structures:—the common nucleus of the abducens and facial nerves with the root-fibres of the abducens, the inferior facial nucleus and the facial nerve, the superior olivary body, and a large portion of the formatio reticularis. The motor portion of the fifth was intact, and so also was the greater part of the sensory fifth nerve. Above the limit of the focus, there was secondary degeneration of the fillet (*schleife*), which ceased at the upper boundary of the pons; below the focus there was secondary degeneration of the olivary body and the fillet. The degeneration of the latter could be traced as far down as the level of the decussation of the pyramidal tracts, the area of degeneration being here situated at the periphery of the lateral columns immediately behind the anterior nerve-roots.

Meyer, in commenting on this case, draws attention to the absence of convulsions and myosis, two symptoms which are generally found in cases of hæmorrhage into the pons. The paresis of the left extremities is attributable to the pressure exerted by the clot on the pyramidal tracts (which were intact): as absorption proceeded, this pressure so far diminished that ataxia of the arm was the only symptom of interference with the tracts that remained. The paralysis of the left rectus internus was not due to lesion of the left oculo-motor nerve, it was probably due to lesion of the fibres of the posterior longitudinal fasciculus, which, as Duval and Laborde have shown, unites the nucleus of the abducens of one side with the nuclei of the trochlear and oculo-motor nerves of the other side.

Tuczek on Spinal Lesions in Ergotism.—Tuczek (*Archiv f. Psych.*, Bd. xiii. p. 99) reports 15 cases of spasmodic ergotism which came under his notice during an epidemic of the malady, all of which presented symptoms of spinal disease. The most frequent symptom was absence of the patellar tendon reflex. This was

observed in every case. Other symptoms were paræsthesiæ, ataxia, diminished sensibility to pain, &c. These symptoms showed themselves months after the acute intoxicant stage, when the patients were in a condition of marked cachexia. In four of the cases (one was only 9 years old, another 16) a post-mortem examination was made, and in all there was found sclerosis of the external division of the posterior columns. Pathologically, the cases differed from tabes solely in the rapidity of the development of the morbid process. The author finds an analogy to the appearance of tabes in ergotism in the occurrence of an acute primary disease of the posterior columns in exceptional cases of pellagra, lepra, absinthe-poisoning, scarlet fever, and diphtheria.

W. J. Dodds, M.D., D.Sc.

Lemoine and Lannois on Spinal Perimeningitis. (*Revue Mensuelle de Médecine et de Chirurgie*, Juin 1882).—A case is related in which paraplegia was rapidly developed five days after a slight fall, attended with symptoms resembling those of acute ascending paralysis; death occurring in this short period, attended with the phenomena of asphyxia. The autopsy showed the rare lesion of softening and disintegration of the spinal cord from pressure of pus formed in the perimeningeal cellular tissue.

A parallel case is quoted by the authors from the *Berliner Klinische Wochenschrift*, 1877, reported by Lewitzky.

Case of Pseudo-muscular Hypertrophy. By Dr. C. A. Pekelharing, Utrecht (Virchow's *Archiv*, Aug. 1882).—This case, in which the author had the opportunity of examining the spinal cord, was that of a boy fourteen years of age. His mother reported him to have been born without any abnormal appearance. Soon after birth the disproportion of the size of his head to the rest of his body was very evident. His growth was tardy. He only learnt to speak at three years of age. At four years his locomotion was imperfect; he limped, and often fell. The calves of his legs were thick; and when he was five years his arms also large, but powerless. His hands and feet suffered from cold. He maintained a half-sitting, half-kneeling posture, the thigh firmly bent to the trunk, and the legs flexed on the thighs. Any voluntary movement was difficult. The muscles, though they felt large, were very feeble; they responded only to a strong electric current. The body generally much emaciated; the thorax deformed. He suffered from disorders of the digestive and respi-

ratory organs. He died from an attack of bronchitis in three days. On post-mortem examination there were found traces of former disease in the lungs and pleura. The cranium was thick and hard.

Examination of the Spinal Cord.—The central canal in the cervical and dorsal regions was dilated and irregular in outline. Nuclei were accumulated, more especially where the canal appeared bent upon itself; in the lumbar region and conus medullaris the lumen was completely occluded by nuclei. On a level with the canal on both sides was a wide opening, which communicated with the anterior spinal fissure and contained a distended vessel which joined the anterior spinal vein.

Throughout the length of the cord the anterior horns were seen to be deficient in nerve-cells, and these only feebly coloured, whilst those in the lateral columns were unaltered. No change was perceptible in Clarke's posterior vesicular column. In some parts the connective tissue was thickened, but not symmetrically. The cells in one section were totally wanting. In the conus medullaris all the cells of the lateral regions of the horns were degenerated. In the grey substance the vessels were numerous and distended, and here the degeneration of the nerve-cells was most advanced.

In this instance, as in those recorded by Clarke, Gowers, and Ross, with the atrophy of the muscles was found degeneration of the cells of the anterior cornua of the cord. The question arises, whether the muscular atrophy is the cause of the degeneration of the nerve-cells, or whether the pathological condition of the nerve-centres is to be blamed for the malnutrition of the muscular fibres. It would seem that the latter is the right answer. In the first place it is not to be supposed that an inflammatory process could spread from the muscles along the spinal cord; neither the nerve-roots, nor the white columns of the cord, show any corresponding softening. The dilatation of the vessels and the abnormal state of the circulation in the grey matter may be assumed to be one cause of the atrophy of the muscles.

Perhaps in the case before us we may go a step further. The patient had been the subject of chronic hydrocephalus. The thickness of the skull left less space for the fluid in the ventricles. The wide lumen of the cord was evidence also of a degree of hydrorachis, the fluid of which had gradually decreased. Were the spinal cord a soft-walled tube, the removal of its fluid contents would cause a collapse of its walls. This not being exactly the

case, the walls of the central canal would yield towards its lumen, and the vessels of the surrounding structures would be affected by the diminished pressure. The vascular canals on both sides of the central canal were extraordinarily large. The hyperæmia hence induced would of necessity influence the nutrition of all adjacent structures—a state of œdema might take place in the grey matter, with a disintegration of the epithelial cells filling the central canal. Thus we revert to the opinion of Duchenne as to the cerebral origin of this form of paralysis. Notwithstanding that many cases of pseudo-muscular hypertrophy have been recorded since Duchenne's time, the presence or absence of hydrocephalus has not been noticed, although in some instances this must have had considerable influence in its causation.

Sarcoma of the Orbital Surface of the Brain, with wasting of the Gyri Recti. By Dr. Otto (Illenau). (Virchow's *Archiv*, Sept. 1882.)—This case, Dr. Otto observes, presents two points of interest: first, the origin and seat of the tumour, and secondly, its relation to the question of localisation. The patient was thirty-seven years of age, of healthy aspect, in December 1879. He inherited no special tendency to disease. Two of his brothers were rather feeble-minded. Himself had the ordinary mental powers. He was married, and the father of three children. Had been an intemperate man. In the summer of the same year, 1879, he had an attack of jaundice with giddiness, &c.: after this he became changed in manner, began to entertain suspicions that he was being poisoned, that his life was being plotted against, that his wife was unfaithful to him, &c. &c. Hallucinations of sight and hearing supervened, and lent force to his suspiciousness, and led to outbreaks of violence. About the middle of March these hallucinations diminished, his health generally improved; he occupied himself in garden-work, but his mind gradually became weaker. This amendment did not last. One day he complained of severe pain on the right side of the head; his conjunctivæ became congested; he declared that the drinking-water was mixed with blood. He became violent, diarrhœa supervened, and he died in about eleven hours.

Post-mortem examination.—The surface of brain was flattened, the veins turgid. On raising the brain an elliptical-shaped tumour was found between the dura mater and the under-surface of the frontal lobes; extending from the foramen cæcum to the sella Turcica. The connections of the connective tissue with the dura

mater were easily broken down with the handle of a scalpel; a turbid puriform fluid escaped from the centre. The measurements were: length, 6 cm. (= 2·36 Eng. in.); breadth, 5 cm. (= 1·96 Eng. in.); depth, 3 cm. (= 1·18 Eng. in.); the under-surface was irregular, occupied the position of the *gyri recti*, and extended backward to the border of the optic commissure. The olfactory nerves could be traced uninjured to the roots. The tumour was united to the cerebral substance posteriorly. Microscopically investigated, the mass was found to consist of round-celled sarcoma. No nerve-cells were to be found in the adjacent cerebral convolutions, but débris, fat and pigment cells.

What does this case teach with reference to the question of localisation? The great degree of pressure exerted on both gyri recti, and exclusively on them, might have been supposed to demonstrate their functions as satisfactorily as any set of experiments.

Hitherto no experiments have been directed to investigation of the under-surface of the orbital lobes, since they are almost inaccessible. The experiments of Ferrier have referred the seat of sense of smell to the *gyri uncinati*, but even this is open to doubt. Two cases are on record, one by Longet, the other by Nothnagel, in which the roots of the olfactory nerve were pressed upon, and the adjacent cortical substance had been absorbed, but in which, nevertheless, the sense of smell was preserved. It was in the present case, for the man was a great lover of flowers and their odours, and was a good judge of cigars. Thus it appears that this complete destruction of the cortex of the gyri recti affords no conclusive evidence as to other functions, but rather indicates that these portions of the brain possess some functions, latent and yet to be discovered.

W. B. Kesteven, M.D.

Sciamanna on the Excitation of the Human Brain. (*Arch. di Psichiatria*, 1882, p. 209.)—The author had the opportunity of applying both faradic and galvanic stimuli to the brain (chiefly through the dura mater) of a man who, in consequence of an accident, had lost part of the right parietal bone.

Excitation of the middle of the ascending frontal convolution produced contraction of masseters and closure of the jaws; of the lower third of the ascending parietal: raising of the left ala nasi and corner of mouth; of posterior central fissure (between ascending parietal and inferior parietal lobe): flexion of arm and raising of eyebrow; of posterior portion of plica supramarginalis (near

inferior temporal convolution): rotation of head to left, movements of orbicularis palpebrarum, of tongue, and eyebrow.

Bechterew on the Functional connection of the Olivary Bodies to the Cerebellum. (Pflüger's *Archiv*, 1882, p. 257.)—The author succeeded in destroying the olivary bodies in animals without opening the spinal canal, and observed the following disturbances of equilibration and motion:—

A. Forced movements, such as rolling on the long axis of the body towards the injured side, with nystagmus, one eye being turned down and inwards, the other up and outwards. When the lesion was of a slight nature: running movements forward, or "manège," or throwing the body backwards.

B. Forced positions: lying on the injured side, or rolled together towards the sound side.

C. Want of equilibration: reeling or impossibility of walking or standing when the lesion is bilateral; titubation and tendency to fall on the injured side, when unilateral.

The author has found that lesions in the neighbourhood of the infundibulum of the third ventricle also produce perturbations of the same nature; and concludes that these parts are in close functional relation with the cerebellum. It is probable that Goll's (sensory) columns terminate in the olives. Meynert thinks that the latter organs are connected by fibres within the medulla with the opposite lobe of the cerebellum; but this view is inconsistent with the fact of the rolling movements occurring towards the injured olive, as they do towards the injured cerebellar peduncle.

The general conclusions are, that we may conceive the semicircular canals, the infundibular region and the olives, as organs transmitting to the cerebellum centripetal influences derived from the acoustic, optic and tactile organs respectively, all of which are necessary to the elaboration of the outgoing equilibrating impulses.

Monakow on the external Acoustic Nucleus and the Restiform Body. (*Neurolog. Centrbl.* No. 21, 1882.)—The author divided the left half of the spinal cord immediately below the decussation of the pyramids in a rabbit on the day of its birth. Six months afterwards the brain was examined, and the following changes found: atrophy of left lateral columns of the medulla; partial atrophy of the left formatio reticularis; atrophy of the lateral cerebellar tract; atrophy of left funiculus cuneatus and

its nucleus; atrophy of the external acoustic nucleus; partial atrophy of the left corpus restiforme (inner side); partial atrophy of cortex of upper vermiform process.

No change in the auditory roots, ascending trigeminus root, or inner part of cerebellar peduncle. Hence the author concludes that the external acoustic nucleus is in relation with spinal fibres, and not with the auditory nerve nor the cerebral peduncle; that the funiculus cuneatus passes partially through the corpus restiforme; that the lateral cerebellar columns terminate in the superior vermiform process.

[In a paper recently read before the Société de Biologie, Laborde gave an account of some experiments made by M. Duval and himself on the semicircular canals and the corpus restiforme. Injury in both cases gave rise to the same phenomena (loss of equilibration). He describes certain fibres which he believes to start from the ampullæ and some of which go to the restiforme body, others to the cerebellum.—*Rep.*]

Lepine on Trismus of Cerebral Origin. (*Revue de Médecine*, 10, 1882.)—The author observed a case of left hemiplegia, with trismus, following hæmorrhage into the claustrum, external capsule, lenticular nucleus, and pressing upon the cortical layers of the insula and foot of the ascending frontal convolution. The trismus was tonic, and lasted the three days which intervened between the attack and the death. No other spasmodic symptom was present. The author thinks that the trismus depended upon irritation at the foot of the ascending frontal convolution, where Ferrier has localised the motor centre of the jaw in the monkey. It is strange, he adds, that the phenomenon is not more commonly met with, but quotes a few cases by various observers where trismus co-existed with lesion in the neighbourhood of ascending frontal.

It may be said in explanation of this fact that the masticatory movements require strong electrical stimuli for their production; mastication, like walking, is a less "cerebral" act than movements of the arm, for instance. This consideration may also explain, perhaps, the bilateral occurrence of trismus in unilateral lesion.

As a help to localising the lesion, trismus can scarcely give a certain clue when accompanied with simple hemiplegia, but can do so when associated with a monoplegia or aphasia.

Eisenlohr on Circumscribed Anterior Poliomyelitis in the Adult. (*Neurologisches Centralblatt*, No. 18, 1882.) The

author describes a case in which a number of muscles of the right arm and hand were degenerated, atrophied and paralysed. Atrophy was present in the interossei; the thenar muscles were atrophied and paralysed. Of the muscles supplied by the musculo-spiral the supinator longus was healthy; the triceps and extensors much atrophied and paralysed. The pectoralis major and serratus anticus were also deeply affected. The deltoid, pronators and flexors of forearm were somewhat wasted, and weaker than normal.

A careful examination of the cord showed it to be healthy, except in the cervical region, when, between the sixth cervical and first dorsal roots on the right side there was evidence of sclerotic changes. These were confined, at the upper part of the altered tract, to the postero-lateral group of large cells. From the level of the lower fibres of the sixth root the whole anterior horn is implicated as far down as the level of the eighth root, the lateral portion being most altered. The white matter was normal, with the exception of the fibres forming the roots.

The author concludes with some remarks concerning the spinal localisation of the motor centres of the arm muscles. The escape of the flexors shows them to be innervated from above the sixth root. The implication of the triceps and pectoralis, on the other hand, proves them to derive their supply from the sixth to the eighth roots. The same remark applies to the extensors of the hand and fingers. The partial paralysis of the hand muscles agrees with the view that they are innervated by the eighth cervical and first dorsal roots. It is, however, difficult to explain the paretical condition of the long flexors and deltoid muscles. Unfortunately no examination had been made with reference to their reactions to the galvanic current.

Vierordt on Atrophic Paralysis of the Upper Extremity. (*Deutsches Arch. f. Klin. Med.* August, 1882.)—The discovery of the influence of the anterior horns upon the nutrition of muscles, gave rise to a tendency to assume poliomyelitis in almost every case where muscular atrophy was a prominent symptom. A reaction in the opposite sense has lately become manifest, and Leyden has severely criticised those views, asserting the frequency of neuritis as a cause of atrophic paralysis. Morbid anatomy is, by itself, insufficient to solve the problem before us. A careful clinical analysis of a large number of cases is required. Nineteen cases are described by the author, who insists upon the importance of four points of view from which to compare the phenomena observed in

each: the chronological relations of paralysis and atrophy; the localisation of paralysis in the various muscles; the presence or absence of concomitant sensory disturbance; the electrical reactions.

Analysis of his cases in the light of these considerations reaches the following conclusions:—

In peripheral paralysis of traumatic origin the motor disturbance is confined to muscles supplied by the injured nerve, and is followed by atrophy. Loss of sensation is present when the lesion is severe, but usually occupies only a part of the district involved. Electrical reactions vary with the depth and date of the lesion; near the injured spot swelling and tenderness of the nerve testify to secondary neuritis.

In peripheral neuritis the paralysis is usually confined to the province of one nerve. Muscles innervated by nerves given off high up by the affected trunk may escape. Paresis precedes atrophy, but not always. Sensation, when diminished, is not extensively so. Reaction of degeneration occurs when the disease is severe, but the responses of the various muscles involved differ greatly. There is frequently swelling and pain along the nerve.

Progressive muscular atrophy affects muscles either in a diffuse, untypical manner, or according to physiological groups, usually beginning in the small muscles of the hand. Atrophy and weakness progress together. Reactions of degeneration usually present, but not easy to demonstrate. No sensory symptoms. Fibrillary contractions frequent.

Anterior (chronic) poliomyelitis attacks physiological groups of muscles. Atrophy secondary to loss of power in ordinary cases, but may follow a parallel course. No evident sensory disturbance. Electrically, partial or complete reactions of degeneration.

A. DE WATTEVILLE, M.A., M.D.

END OF VOL. V.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET AND CHARING CROSS.